THE HUMAN SPINAL COLUMN AND UPWARD EJECTION ACCELERATION: AN APPRAISAL OF BIODYNAMIC IMPLICATIONS

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Foreword

The critical review of the literature which forms the basis of this report was conducted under Project 7231, "Biomechanics of Aerospace Operations," Task 723101, "Effects of Vibration and Impact." The work was conducted in the period July 1965 through June 1966.

Publication of this report does not constitute Air Force approval of the report's findings or conclusions. It is published for the exchange and stimulation of ideas.

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Abstract

Vertebral compression represents a significant percentage of the morbidity associated with upward ejection. Vertebral and intervertebral structure reacts to and is sometimes irreversibly altered by ejection acceleration. Design and material properties of the normal vertebral column are sufficiently constant that when structural characteristics are defined and acceleration profiles known, prediction of failure may be made. Compressive load analyses of vertebra-disc complexes have demonstrated that the vertebral end-plates are the initially failing structures of the spinal column. From experimental data on vertebral breaking-loads, acceptably accurate probability-of-injury curves for static loading have been generated. These data together with data describing the dynamic response characteristics of the human body permit calculation of the probability-of-injury for dynamic loading produced by exposure to impact accelerations. As an aid to the designer of ejection systems, application of these concepts should refine the estimate of "safe" acceleration profiles and minimize the risk of irreversible vertebral deformation.

SECTION I.
Introduction

With the advent of aircraft ejection seat escape systems 25 years ago, injury to the spinal column began to represent a significant percentage of the morbidity and mortality associated with this mode of aircraft-pilot separation.

Before World War II, the Germans, anticipating crewmember escape difficulty from high performance aircraft, initiated the development of ejection systems. Their engineering research and development program actually began about 1939. Junkers made the first installation of an ejection seat in 1941. This early seat was a ballistic catapult system which had not been extensively tested. In the early 1940's independent research and development were initiated by Heinkel and Dornier aircraft development engineers. From 1941 on, ground rigs were incorporated and test seat firings were made using both dummies and humans. Early reports on the human tests revealed a significant incidence of fractured vertebrae. The early German developers were also concerned about a painful downward "snapping" of the head, neck, and shoulders. In many of those early development studies, restraining harnesses were not used. By 1944 the Germans had reported on 40 dummy ejections from aircraft and also three successful human ejections by experienced parachutists. By the end of the war they had operationally used an ejection system 60 times.

British ejection system development had been planned earlier, but it was not until 1944 that the Martin-Baker Company instituted a comprehensive, planned program encompassing engineering development and human response testing. This program's investigators explored subjective physiologic responses in an attempt to gain some idea of tolerance limits in a somewhat unsophisticated manner, but one that was an improvement over the earlier German studies. The British observations of vertebral fractures led to development of a two-staged catapault that permitted more controlled vertebral loading with subsequently less probability of vertebral injury. To avoid the painful head and neck flexion, the British incorporated the idea of "blind firing." By using a properly positioned overhead "curtain," pulled down over the face and held against the upper chest, the ejection sequence was initiated in a manner that prevented upper body flexion.

By 1946 Sweden had developed an operational ejection system and the British had perfected a relatively dependable, fairly sophisticated Martin-Baker seat. This system, with various modifications and innovation, has been incorporated into many of our own and allied aircraft during the past two decades.

In the United States ejection system design began in July 1945. The development program was characterized by an engineering phase for design of a compact catapult-seat complex. Additionally, however, there was a comprehensive "aeromedical" phase to delineate human ejection tolerance. Both phases aimed at development of a system to safely clear a man from an aircraft traveling 600 miles per hour. In July 1946 the first U. S. human test from an aircraft was successfully carried out at Wright Field.

Over the years numerous advancements in both aircraft and escape systems have occurred. Propellants specifically designed for escape system use were introduced into the catapult gun so that the acceleration-time history could be kept within physiological limits. Automatic devices were invented to assure man-seat separation and parachute deployment. These were of im-

measurable benefit in saving pilots rendered unconscious during escape or held to the seat by spin. As more and more ejections occurred, it became obvious that a system was necessary that would permit pilot clearance from an aircraft that was "in trouble" before it left the ground. This requirement was met by Navy development of a rocket-assisted catapult sequence. Using this system, the man is cleared from the aircraft by a ballistic propellant catapult. A rocket then "kicks in" and provides a lower but more sustained upward acceleration that enables the seat-man complex to obtain an altitude at which seat separation and chute deployment automatically occur.

As higher and higher speeds have become attainable with jet propulsion advancement, two difficult problems have arisen. At 0.85 Mach-plus airspeeds, intolerable acceleration profiles become necessary to clear a tail fin. Also at these aircraft velocities, sudden exposure to windblast is devastating. Partial solutions to the two problems have been met through development of downward ejection systems and capsule escape systems. Additional advantages of these capsule or "pod" systems are pressurized environment, oxygen and survival equipment, restraint during deceleration, and flotation capability upon landing.

Out of painfully accrued, extensively detailed, operational ejection tabulations, investigators have come to realize that no matter how sophisticated the system, the highest percentage of survivals occurs when a crewmember is able to manipulate his failing aircraft to optimal orientation and airspeed-altitude conditions.

In the present era of high performance aircraft and space exploration, the problem of accelerative spinal injury persists. However, the effort to design escape systems that will minimize trauma to the vertebral column has become increasingly complex.

This report represents an attempt to bring together the collected information on those factors that influence safe ejections. It is an effort toward a unified concept outline for the aerospace system designers, specialists in biodynamics, and flight medical officers, all of whom are intimately associated with the overall problem.

For any escape system sequence in which certain stresses combine to exceed human tolerance, there is a significant probability of micro- or macrovertebral column injury. To a great extent this type of injury is preventable. Fortunately, when it does occur, localized symptoms usually herald its presence. Methods are available to document its presence. Aerospace literature contains little describing the anatomic specifics of vertebral fracture incurred as a result of exceeding structural tolerance. Even less has been documented about the dynamic relationship between the discs and the bony spine during ejection. Nothing is available which accurately describes the long-term sequelae of spinal column injury. Reports have documented minor spinal injuries without defining precisely what constitutes minor injury. Herein an attempt has been made to correlate the clinical complaint with the structural alteration. The precise anatomic alterations that occur within the vertebral column during ejection are explained and exactly how these sequential alterations are influenced by the design and use of the system are discussed.

This report deals primarily with the vertebral body and intervertebral disc response during ejection. Fracture, dislocation, posterior arch disruption, and cord transection, which also occur, are not discussed here, because these injuries are usually associated with an unsuccessful (fatal) ejection sequence. As more complex escape systems and landing vehicles become operational, force orientations upon the vertebral column will be more variable. However, even with the biodynamic implications of severe body twisting, rotation, and flailing, during which other injuries may occur, compression spinal fracture probably always will be an escape system problem. I

believe, as will become apparent, that accepting the accuracy of spinal biomechanics, documenting safe accelerative characteristics for a reliable ejection system, and appreciating the occurrence of unsuspected underlying biologic variability; I may comfortably state that a great portion of ejection-incurred spinal injuries result from abnormal ejectee posture, unsuspected congenital spinal weakness, and dynamic spinal overloading occurring secondary to either "overshoot" or improperly arranged restraint and stabilization systems.

Subsequent to all of the innovations, modifications, and adaptations that characterize modern escape systems, unchanging man tolerates no more acceleration trauma in the caudal-cranial axis than was grossly postulated 25 years ago. Design capability continues to be limited by biologic breaking points. The challenge is to precisely define and utilize these end points to their maximum benefit.

SECTION II.

Spinal Column Anatomy

The human spinal column is structurally composed of a bony, cartilaginous, and ligamentous complex *(figure 1)* that flexibly supports the torso and protects the spinal cord. A total of 33 bony vertebral elements are separated by fibrocartilaginous intervertebral discs which, along with vertebral joint capsules and ligaments, serve to stabilize successive vertebrae. Each vertebra *(figure 2)* has an anterior weight-bearing portion, the vertebral body, and a posterior arch, which shields the spinal cord and serves as an attachment for the paraspinous muscles. Five of the fundamental 33 vertebrae are fused into a sacrum and another 4 form the coccyx or "tailbone." The remaining 24 vertebrae are positioned successively as 7 cervical, 12 thoracic, and 5 lumbar. The vertebral bodies are interconnected by strong longitudinal ligaments that extend along the anterior and posterior surfaces from the sacrum to the skull. The posterior vertebral arches are also strongly bound to one another by three specialized elastic ligaments, as illustrated in *figure 3*. At birth, individual vertebral bodies are immature and only scantily ossified. During the first few years of life vertebral growth and maturity rapidly progress. At both the superior and inferior aspects of the vertebra, cartilaginous collars, which represent epiphyseal growth rings, become apparent. Before the age of 6 or 7, two bony plates, the *vertebral end-plates*, develop on both the cephalic and caudal surfaces of each vertebra. During an individual's childhood and part of adolescence, blood vessels perforate these anatomically distinct and structurally important end-plates. By adulthood, however, these vessels no longer function and fibrocartilaginous tissue obliterates the original channels. Nonetheless, these obliterated channels structurally persist as nonosseous areas of fundamentally osseous vertebral body components. As an individual advances from infancy through youth and into adult life, the vertebral body bone between the end-plates matures. Histochemically, this spongy-type osseous tissue is composed of a mineral apatite dispersed throughout a protein collagen matrix. Apatite is characterized by relatively high compressive strength and stiffness, but a rather low tensile strength. Contrariwise, protein collagen has relatively low stiffness, but high tensile strength. Together these inorganic and organic components yield a material with good elastic characteristics and relatively high compressive-tensile properties. As bone ages, elasticity increases, tensile strength changes little, and compressive strength decreases (Stech, Jan. 63). Older vertebral bone is also characterized by lower stiffness values. Although it is tempting to attribute such diminished stiffness and compressive strength to the decreased amount of compact bone present in older specimens, other more subtle, nonapparent, age-dependent, biochemical changes are also occurring. The altered strength characteristics of older vertebrae are probably a result of changes in both of these dynamic processes.

Fibrocartilaginous intervertebral discs are positioned between successive vertebral bodies *(figures 4, 5)*. Individually variable in thickness, these structures constitute from one-fourth to one-third of the total spinal length. Most anatomists describe the structural makeup of the discs as consisting of two components, the annulus and the nucleus. In reality, however, and as described by Hirsch and Nachemson (1954), each disc is made up of three distinct, though anatomically combined parts. The annulus fibrosis is composed of concentrically layered fibroelastic tissue. Both superiorly and inferiorly, disc annuli are firmly attached to the adjoining vertebral bodies. The anterior-longitudinal ligament is firmly bound to the annulus, while on the dorsal surface the posterior longitudinal ligament is less firmly anchored. The fibrotic annulus is thicker in the anterior area as compared with the posterior area. Within the annulus lies a second integral component of the disc, the nucleus pulposus, a watery gel composed of dense collagen permeated

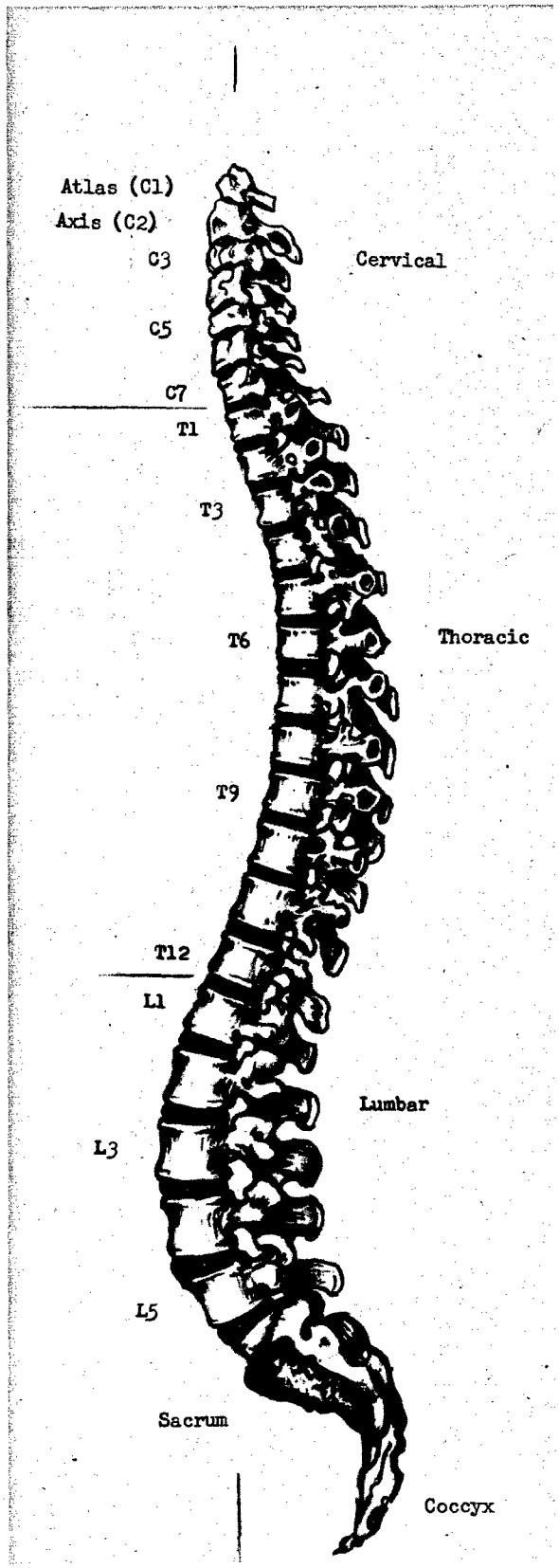


Figure 1. Isolated human spinal column. The cervical and lumbar curves of the correctly postured spine are convex anteriorly whereas the thoracic curve is convex posteriorly.

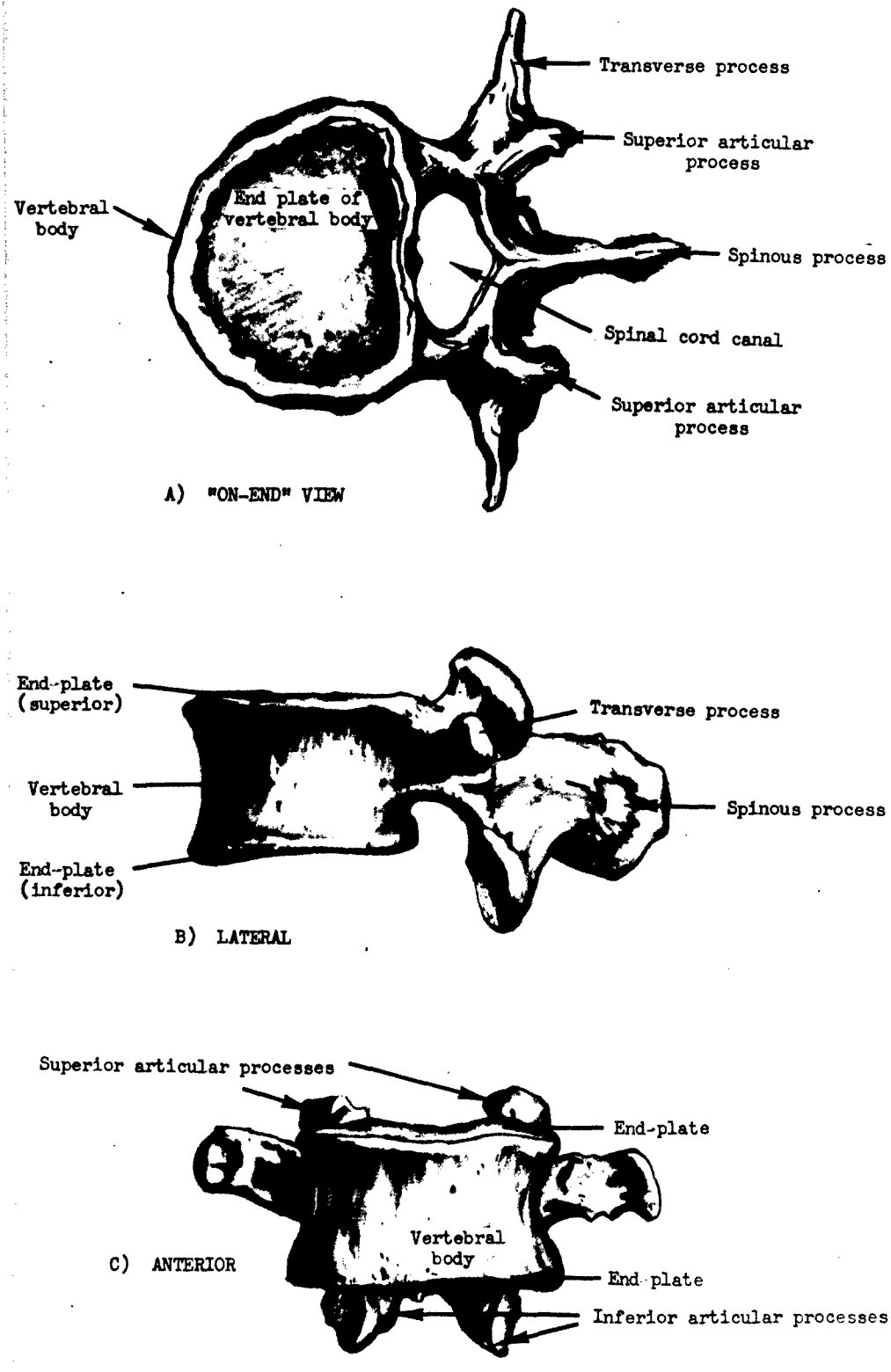


Figure 2. Views of Vertebral Body

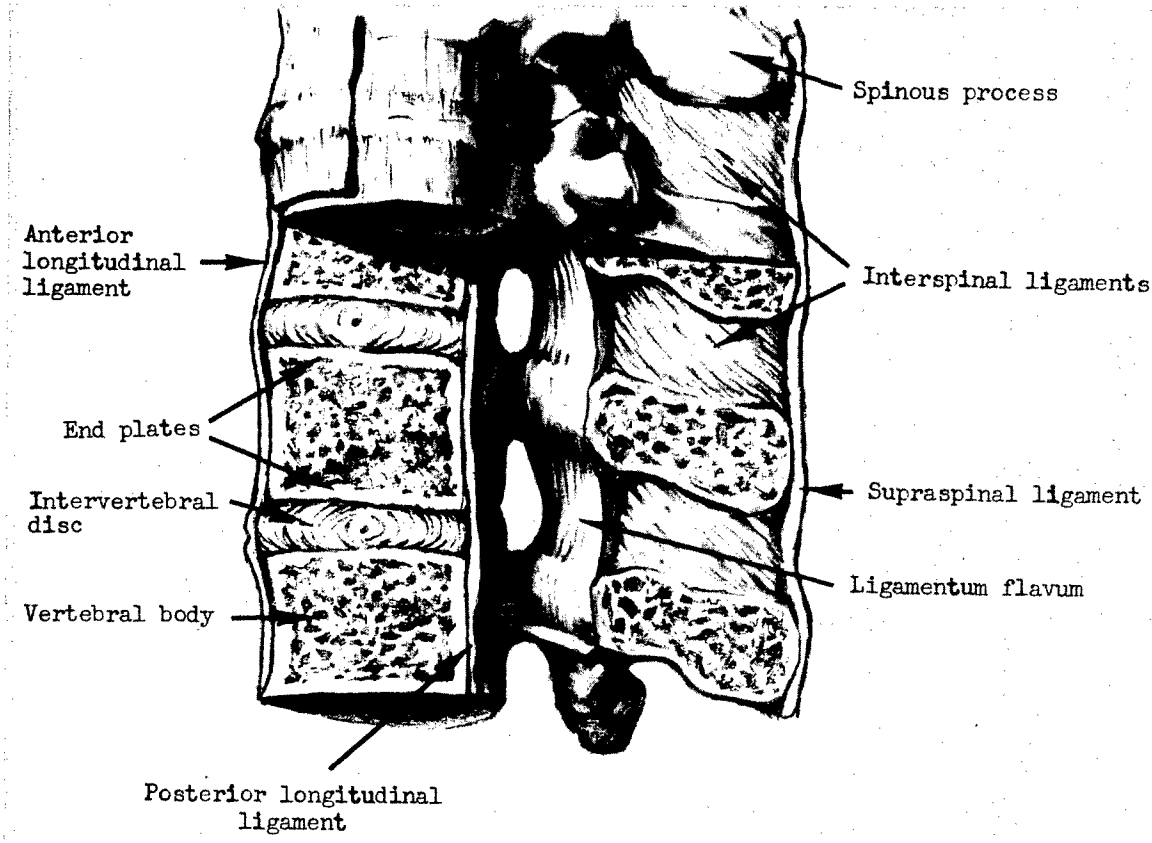


Figure 3. Anatomy of Intervertebral Stabilization
(Each vertebra has an anterior, weight-bearing portion, the vertebral body, and a posterior arch which shields the spinal cord and serves as an attachment point for the powerful back muscles. The anterior and posterior longitudinal ligaments are inherently attached to the vertebral bodies and the discs. As illustrated, the posterior arches are also firmly attached to one another by specialized elastic ligaments.)

with polysaccharides and containing a significant proportion of chondroitin sulfuric acid. This part of the disc, which is supposedly capable of absorbing 16 times its own weight in water, is not positioned in the anatomic center of the annulus. It is slightly posterior but designed to align with those areas of adjacent vertebral end-plates which represent the central pressure points of successive vertebrae. The third anatomic entity of each disc is a pair of fine hyaline cartilage plates which are derived from the annulus and form borders between the nucleus of a disc and the osseous end-plates of adjacent vertebral bodies. As an individual matures and advances into adult life, the semifluid consistency of the nucleus changes into a more solid substrate. With the continued wear and tear of advancing years and concomitant with the above-mentioned vertebral body structural alterations, nuclear solidification progresses.

The entire supporting spinal column, vertebrae and discs, is embryologically formed out of and around a primitive "tube" that early in fetal life is composed of pleuropotential elements. Because congenital abnormalities occur, arrest or overgrowth at any point during the differentiation and development of the vertebral column may result in structural defects. Such defects or malformations are extremely important when they occur in a pilot population. Appreciation of their occurrence and ability to recognize them is an essential part of individual spinal evalu-

Figure 4. Schematic Illustration of an Isolated Intervertebral Disc

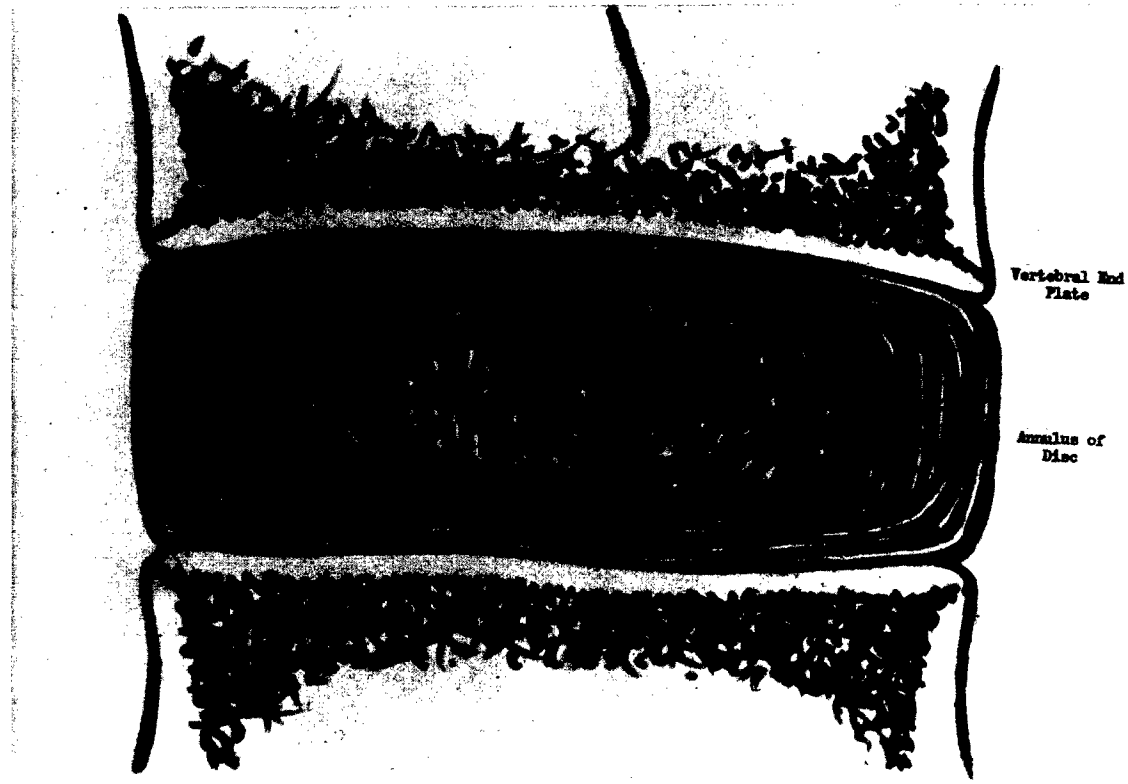


Figure 5. Intervertebral Disc

ations. Partial persistence of the primitive embryological tube may occur and be present in some individuals without clinical evidence of its presence. If this tube persists between two discs, the vertebral body is centrally lacking and the nucleus pulposi of two adjacent discs may be in contact with each other. Similarly, tube remnants within a vertebral body can cause defects without affecting the integrity of the end-plates. Although Schmorl (1931) believes that it is difficult to pick up such developmental errors on routine roentgenographic films, he has warned that discs which bulge into vertebral bodies in young people suggest weakness. In addition to such tube defects, posterior arch anomalies occur. A central cleft in a posterior process of an arch is not an uncommon occurrence which, dependent upon size, may never cause difficulty. Hemivertebrae also occur as do vertebral alignment errors. Approximately 30% of the population have x-ray detectable abnormalities of the spine that for the most part remain asymptomatic. Their potential significance in a pilot population is apparent. Although the recognition of such anomalies will be more fully elaborated in a later section, the reader should know at this stage that both detectable and "silent" structural spinal variation do occur. Obviously, the supporting characteristics and capabilities of such vertebrae, depending upon the extent of the defect, may be less than those of their normal counterparts.

SECTION III.

Physiology of the Vertebral Column

The human vertebral column is constructed in such fashion that it provides flexion, extension, lateral bending, rotational capability, and stability to the human skeleton. The normal adult column has four predominant curves. The cervical and lumbar curves are convex anteriorly while the thoracic convexity is directed posteriorly. The concavity in the sacral region is anteriorly directed. Such an alternating convex-concave arrangement results in greater elasticity for the transmission of body weight or added loads. It also results in superb positional flexibility and a greater facility for dynamically maintaining the center of gravity which, for upright man at rest, lies just anterior to the sacral promontory. Carey (1928) estimated that a normally curved spine is approximately 16 times stronger than it would be if the vertebrae and discs were arranged in a straight line. Actual vertebral joint motion is dependent upon the posterior arches with their ligaments, the anterior and posterior longitudinal ligaments, and the annulus-nucleus complexes of the intervertebral discs. Such movement between vertebrae takes place in the resilient intervertebral discs and at the joints of the posterior arch articular processes. Although actual displacement between vertebrae is relatively small, total column motion is considerable. Keller (1955) noted that the total area of an intervertebral disc is directly related to its particular position in the vertebral column. Hence, the thoracic vertebrae, which experience relatively limited motion, have smaller discs, whereas the cervical and lumbar segments of the spine, which experience freer mobility, have larger discs. End-plate pressure is distributed partially over the annulus and partially over the nucleus of the intervertebral disc. Pressure absorbed by the normal nucleus is hydrostatically distributed over both the adjacent end-plates and the internal aspect of the annulus. The changes a disc undergoes during life alter its functional properties. Whereas the moisture content within the nucleus of a newborn child is approximately 88%, it diminishes to about 68% in an elderly individual. Subsequent to such change, disc mobility is reduced and pressure transmission to adjacent vertebrae changes in the manner in which it occurs. Disc degeneration per se occurs subsequent to both decreasing nuclear fluid content and the simultaneously occurring everyday wear and tear of constant load stress and strain. Both annular bulge, and its later prolapse-initiating defect, annular tear, are often partially or wholly related to nucleus pulposus turgor loss. A cross section of a degenerated disc from a senile individual usually reveals a hard, dry, yellowish surface. Although perhaps physiologically normal for its age, mobility in such a disc is reduced. The greater the degree of nuclear dehydration and degeneration, the greater is the proportion of the pressure that must be supported by the annulus.

When an adult human male moves from a reclining to an upright position, lower vertebral column disc nuclei are subjected to loads that average 45 kg. If this same individual bends or extends his spine, as is often done when one stretches, this same nucleus must support from 100 to 130 kg. When the body is bent forward at a 90° angle, the pressure transmitted to the lower lumbar discs is about ten times as great as the weight being lifted or supported. The implications of annular strain and vertebral body loading occurring under these conditions and in the presence of a degenerated disc are apparent. A poorly postured spine undergoing accelerative forces experiences the same type of load distribution, but in a dynamic manner.

The normal young (20-40 years) spine is capable of generous mobility, flexibility, and loading support. It is also capable of good recovery from injury in the absence of simultaneous spinal cord damage. With proper posture and sensible load onset rates, the healthy spine will support remarkable static and dynamic forces. However, if either of these factors are ignored or insufficiently regarded, vertebral body or intervertebral disc injuries can be anticipated to occur with relatively high frequency.

SECTION IV.

Spinal Biodynamics During Ejection Acceleration

SPINAL STRUCTURE UNDER STATIC MECHANICAL LOADING

Biomechanics is the investigation of mechanical phenomena that transpire in living organisms. Biodynamics describes the effects that various force environments have on biologic systems. Any fixed object responds to external environmental forces by the development of internal stresses which, if of sufficient magnitude, will alter the structure, form, and functional capability of the object. More simply stated, externally applied force results in internal structural strain to a point where failure ultimately takes place. Damage to biologic tissues from the forces resulting out of acceleration must take place in essentially the same manner that damage to nonbiologic structures occurs as a result of such forces. If the structural characteristics of a biologic material can be defined, and the magnitude and acceleration of a particular force applied to that material can be described, one should be able to predict whether or not failure will occur. In the investigation of material strengths, different types of applied force and different types of failure can be described. The primary concern in this report is accelerative forces directed parallel to the spinal axis. Such forces result in compressive stresses arising within the structural components of the spinal column. Failure of the vertebral column occurring during ejection is a direct result of such compressive stresses. Definition of compressive failure is dependent upon stress-strain (load-deflection) curves. *Figure 6* is representative of such a curve. Stress is represented along the ordinate and strain along the abscissa. Along the compressive response curve are points representative of degrees of structural deformation. The curve departs from a straight line at a point beyond which relatively small force increments cause rather large deflection increments. With departure from this straight line, or linear relationship, a region of initial failure for the material is being approached. This graphic point, beyond which compressive strain is no longer proportional to the loading force, is known as the *proportional limit.* Proportional limit value defines elastic capability and is a representative estimate of the point at which a material begins to fail but is able, upon force cessation, to recover its preload form free of structural damage. Another definition of the limit of elastic response is represented by the *yield point* of a load-deflection curve. Yield point value defines irreparable damage and is a representative estimate of that point at which a material is permanently altered in form and will not recover its original shape subsequent to load cessation. The material is, however, still capable of load support at this point and therefore able to continue its particular function. With increased compression beyond the yield point, total failure will eventually occur. *Total failure* defines that point at which a material crumbles or crushes to the extent that structural integrity is completely destroyed.

Statistical variability is inherent in the mechanical properties of all materials. This variability is even greater for biologic materials, which are continually undergoing dynamic biochemical change. Therefore, strength analysis performed on tissues like cartilage and bone will present variable results. If, however, analyses are performed on a number of specimens from individuals of the same sex, age, weight, general build, and degree of health, the results should follow the normal, bell-shaped curve.

The stress-strain curves generated from biologic materials differ from those generated from many of their nonbiologic counterparts. The latter frequently exhibit more linear load-deflection characteristics over a greater part of their curves. The curves for biologic materials, however, are often more nonlinear. In order to avoid lengthy, complex mathematical analysis linear approximations are usually made, which tend to set certain limitations upon the validity of calculated results.

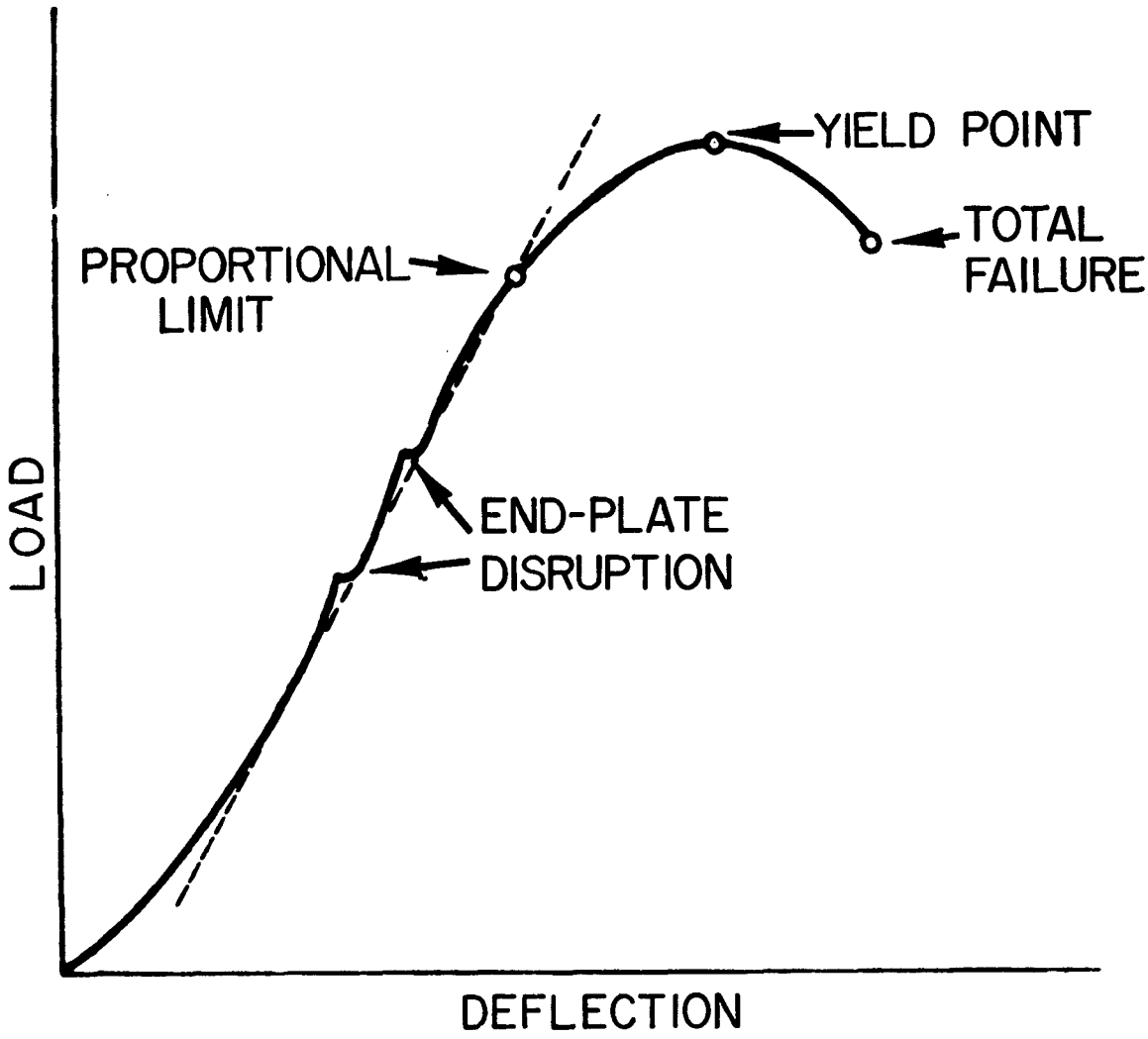


Figure 6. Schematic Load Deflection Curve Illustrating Alterations in Vertebral Body Structure During Compressive Stress

Much of the stress-strain data for the spine has been obtained from human cadaveric material. This is not at all unrealistic if one works with fresh, healthy specimens and maintains, as closely as possible, biologic temperature and water-mineral environments. Another method for obtaining data is to extrapolate animal data to man, which by and large is fraught with hazard, and, since this report deals with data on human subjects, will not be discussed further than mentioning it as a method. Material strength analyses have been performed on human spinal columns. If those data yield valid compressive breaking load levels for spinal discs and vertebra, living humans who exceed the levels can be anticipated to incur injury. Similarly, if such derived data are accurate, injury should not occur if human seat-to-head spinal acceleration is kept below the permissible levels.

Turning now to injury modes in vivo, it is p rudent to make a point concerning tolerance and tolerance levels. Subjective tolerance and tissue injury should be separate concepts. Here we are interested in tissue injury. Human tolerance levels for spinal axis ejection forces should be interpreted here as those levels just above which irreversible damage will occur within the structurally most susceptible component of the spinal column.

The mechanism of absorption of compressive forces by the vertebra-disc complexes of the spinal column is fascinating. Both Brown, et al (1957) and Roaf (1960) have reported excellent investigations on the relationship between disc and vertebra biomechanics during axial stresses. They noted that early during column compression there is a decrease in disc volume. This decrease ranges between 1 and 2.5 cc before one of the adjacent vertebral end-plates fractures. Recognizing the fluid retaining capability of the disc annulus and the structural porosity of the end-plates, these investigators believed that this diminishing disc volume resulted from two sequentially occurring events. Early during significant spinal compression, sinuses, fissures, and micro spaces, normally present in all adult discs, collapse. The diffusible intradisc fluid displaced during this process is "pressured across" the vertebral end-plates, which are more porus than the annulus. With continued compression, end-plate bulging occurs and the fluid within the cancellous bone of the vertebral body (both endogenous and that transposed from the discs) is forced out into perivertebral sinuses and veins. With continued spinal compression beyond the energy dissipating capability of this fluid transfer mechanism, vertebral end-plate bulge increases to the point of fracture. An individual may demonstrate this fluid-transfer mechanism in himself. The body height difference that is noted between early morning and late evening measurements is a result of this pheonmena. During the night, when the spinal column is relieved of its load, this fluid is replaced and a subsequent increase in body height occurs.

Roaf and Brown et al also noted that only after end-plate fracture did compressive vertebral body damage occur. They emphasized that whether a disc was histologically "normal" or "degenerated," the same sequence of events occurred. Neither discs with fragmented nuclei nor those specimens with annular bulge secondary to early fibrous tear, "prolapsed" prior to end-plate fracture and vertebral body compression. These investigations show that the precise mechanism of energy disssipation across the disc varies with the age and condition of these specimen.

Brown (1957) documented unsymmetrical annular bulge during compression of older specimens. This bulging repeatedly occurred in the strongest area of the disc. If such compressive bulge were occurring as a result of intradisc hydrostatic effects, (which we know do participate in energy dissipation for young healthy discs) he believed the bulge should have been most marked in the weaker posterior and posterolateral areas of the annulus. The fact that it was most pronounced at the strongest anterior area would be against a primarily hydrostatic mechanism and in favor of direct compression of the annulus itself. There is clinical supporting evidence that intervertebral disc annuli are in themselves capable of support and energy dissipation. Schmorl's node is an eponym for an x-ray evident nucleus pulposus which ruptured through an end-plate into a vertebral body where it became ossified. Its "enucleated" damaged disc, however, still maintains significant intervertebral space. During surgical intervention for disc "herniation" a fair amount of extruded material may be found with a minimally altered disc space. In each of these situations there is a loss of nucleus with minimal if any change in intervertebral space. The only structure capable of maintaining this support is the annulus of the disrupted disc. It would appear, therefore, that although the nucleus potentiates intervertebral disc support and energy dissipating capability, it is not a requisite for either of these functions which may be taken over by the annulus. In summary, the water content of a disc

nucleus results in more efficient energy dissipation and permits more severe accelerative forces. With disc aging and degeneration, however, nuclear water content diminishes and the older worn out nucleus becomes fibrotic and later fragmented. Therefore, in a normal disc, internal pressure is hydrostatically distributed to both annulus and vertebral end-plates. This results in an intervertebral pressure transfer (energy dissipation) by a highly efficient disc using both nucleus and annulus. In the degenerated disc, however, characterized by depleted fluid content, a greater proportion of energy dissipation must be absorbed by the annulus.

Significant contributions have been made on the biomechanics of the isolated disc-vertebra components of the spinal column. These began in 1940 with Ruff who was interested in the breaking strength of vertebrae under axial compression. He was similarly interested in the portion of body weight supported by the most heavily loaded vertebra and the acceleration-time history of compressive vertebral failure. He also investigated what he termed "ultimate spinal compressive strength." Ruff recognized this ultimate strength to be dependent upon the deformability of the most heavily loaded vertebrae, the compressibility of the shock-absorbing intervertebral discs and the elasticity of the upper body. In subjecting fresh cadaveric vertebral specimens to static compressive loading, Ruff calculated breaking strengths from the point on the load-deflection curve at which the first peak occurred. Recall that this represents the yield point beyond which irreversible deformation occurs. Considering the height-maintaining and weight-supporting functions of vertebrae, this type of structural failure documentation is clinically and biodynamically significant. After testing a number of vertebra-disc complexes, Ruff became aware (as Roaf [1960] and Brown et al [1957] later confirmed) that forces were mainly absorbed by the vertebral body which always broke before the adjacent disc incurred discernible damage. Realizing that individual vertebral body energy absorption during acceleration is dependent upon the amount of preacceleration body weight supported by that particular vertebra, Ruff set out to ascertain the percent of total body weight supported by the individual vertebral bodies. He placed subjects on tables designed for minimal table-man friction and, using a "loading yoke," axially compressed the spine in 10-kg increments. The spine was x-rayed after each load increase until a total of 60-kg compression was in effect. The unloaded subject was then filmed in the erect position after which loaded-unloaded intervertebral space differences, measured from the x-rays, were used to calculate individual vertebra strain.

Table I presents Ruff's data on breaking loads for T8 to L5 vertebrae and also his calculated percent-of-body-weight supported by these successive vertebrae. Extending his experimental breaking strength and percent body weight supported data, Ruff derived maximal and minimal G-load tolerances for individual vertebrae by assuming that all specimens were representative of those tested from the spinal column of a 75-kg man. These values and his formulas are presented in table II.

The final portion of Ruff's investigation dealt with acceleration-time-histories. He concluded that for exposure periods of 5 msec to 1 sec, structural tolerance is determined by the static compressive strength of the vertebra most easily traumatized by such loading. For accelerative periods lasting less than 5 msec, structural tolerance is determined by the dynamic strength of the most susceptible vertebra. Dynamic strength of the most susceptible vertebra is a function of both total column disc compressibility and upper body elasticity. *Figure 7* illustrates the "G-time" tolerance levels derived by Ruff

The second significant biochemical investigation of the spinal column was that which Perey of Sweden published in 1957. His analytic methods differed from those of Ruff in three ways: he emphasized "proportional limit" instead of "yield" points; he did dynamic as well as static

TABLE I

BREAKING STRENGTH OF VARIOUS VERTEBRAE

Vertebra	*Breaking Strength (kg)*	*Average (kg)*	*Average (lb)*	*Body Weight Supported (%)*
T-8	640, 540, 609	593	1315	33
9	610, 720, 700	677	1493	37
10	800, 660, 770, 730	740	1632	40
11	750, 720, 860, 755	771	1700	44
12	900, 690, 800, 800	797	1757	47
L-1	720, 840, 900, 800, 800	812	1790	50
2	990, 800, 830	873	1925	53
3	900, 940, 1100	980	2161	56
4	1100, 900, 950	983	2168	58
5	1020, 1000, 1200	1073	2366	60

TABLE II

CALCULATED VALUES FOR A BODY WEIGHT OF 75 kg

Vertebra	*Max Breaking*	*Min Breaking*	*%*	*Max G**	*Min G**
T8	640	540	33	24.9	20.8
T9	720	610	37	25.0	21.0
T10	800	660	40	25.7	21.0
T11	860	720	44	25.1	20.8
T12	900	690	47	24.5	18.6
L1	900	720	50	23.0	18.2
L2	990	800	53	23.9	19.1
L3	1100	900	56	25.2	20.4
L4	1200	900	58	24.3	19.7
L5		1000	60	25.7	21.2

*Using the formulas

$$n_{max} = \frac{P_{Br}max}{\epsilon \times G} \times 100-1 \text{ and}$$

$$n_{min} = \frac{P_{Br}min}{\epsilon \times G} \times 100-1$$

Wherein P_{Br} max and P_{Br} min are the highest and the lowest breaking loads noted in initial testing; ϵ is the percent of body weight carried by the individual vertebra; G is the body weight.

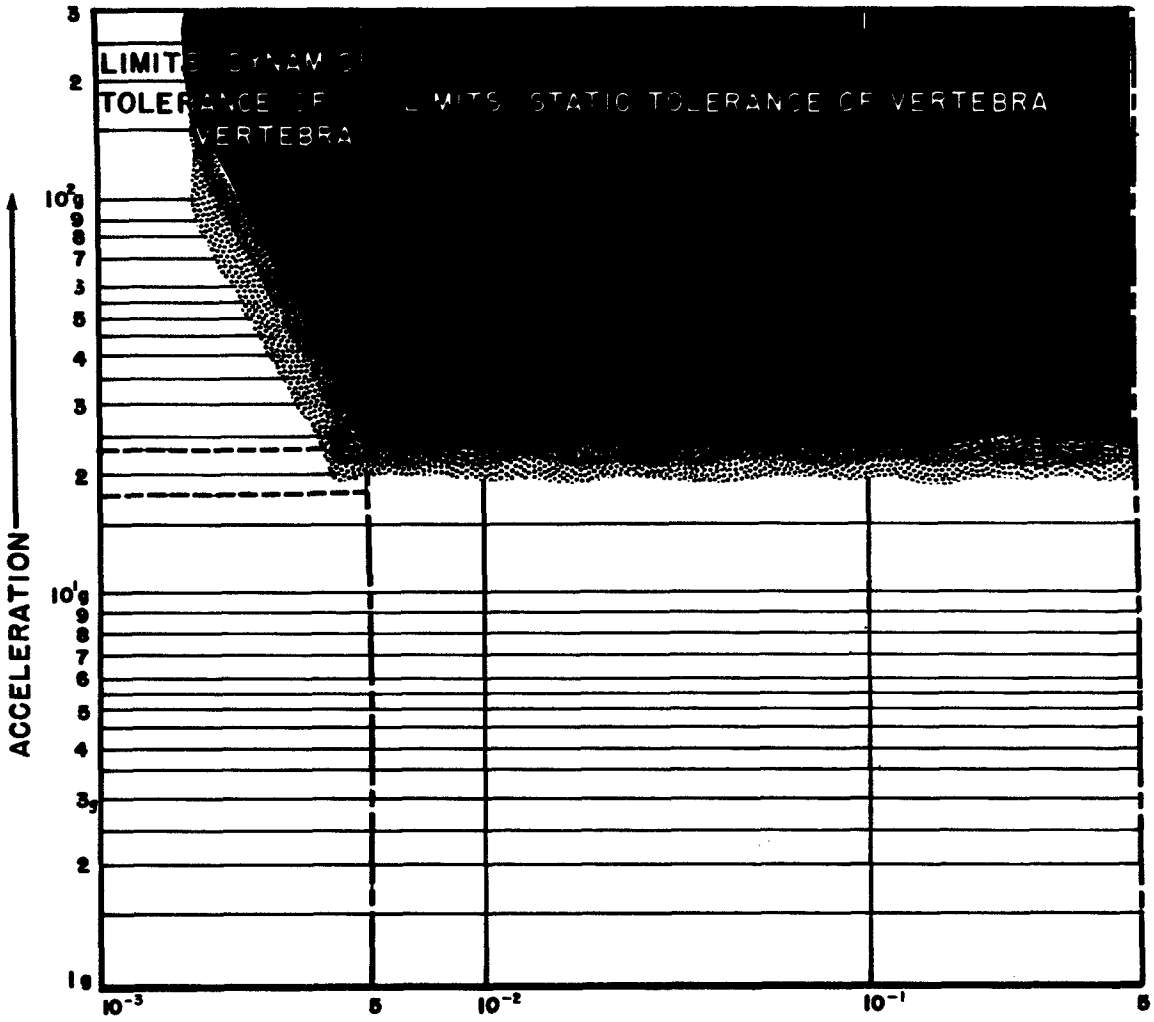


Figure 7. Effect of Acceleration in the Direction Seat-to-Head (—Gz)

loading; and his specimens were more exactly representative of specific anatomic entities than were Ruff's. In emphasizing proportional limit, Perey's results were lower than Ruff's. Although they would appear to be safer by virtue of the definition of proportional limit, i.e., reversible deformation; Perey noted that fracture of the anatomically distinct vertebral end-plate occurred at levels even below the vertebral body proportional limit. Such end-plate fracture represents a subtle event. Pathologically, however, it is an extremely important anatomic failure.

From a structural analysis viewpoint, testing of clean two- and three-vertebrae specimens with discs still attached, as carried out by Perey, should more specifically delineate the structurally most susceptible components of the spinal column. It is unfortunate that Perey's investigation was limited to segmented T12-L5 vertebral complexes. However, a great deal of significant information about the lower spinal column was nonetheless realized. By injecting radiopaque media into discs of test complexes and then taking roentgenograms of dynamic compressive alteration, Perey was able to document the "weakest links" at the moment of damage. His ability to

accomplish this was facilitated by x-ray equipment implemented with image intensification and 48-per-second exposure capabilities.

During one group of dynamic loadings to the proportional limit, Perey identified 20 instances of vertebral end-plate fracture as compared to only 6 instances of irreversible vertebral body compression. He was sufficiently interested in these end-plate fractures to describe three types: centrally situated defects, peripheral "chip" fractures, and fracture fissures that extended across the diameter of the end-plate. Perey warned that many of these end-plate fractures could not be visualized on x-rays that were experimentally analogous to the routinely obtained views in the clinical situation. Many of these x-ray "misses" were documented rather easily, however, by discography and laminography. Subsequent to his dynamic testing, Perey investigated static loading of two- and three-vertebra complexes.

In the 40 two-vertebra specimens subjected to static compression, a definite relationship between age and proportional limit was noted. For vertebrae over age 60, average breaking strength was 425 kp while the average for the group under age 40 was some 40% greater at 780 kp. End-plate fractures in the static test two-vertebral complexes were microscopically evident in 13 instances (32%). Following his preliminary dynamic and static tests, and appreciating the significance of breaking point differentials between end-plates and vertebral bodies, Perey was naturally interested in comparing these two sets of values. Tables III and IV present average results obtained. In this portion of his investigation Perey also ascertained that vertebral bodies compress an average 16% of their total height before the proportional limit is reached. Realizing that the actual vertebral body fracture value lies closer to yield-point breaking loads, more than 16% reversible compression probably occurs before vertebral body "fracture."

TABLE III

LUMBAR VERTEBRAL BODY RESISTANCE WITH RESPECT TO AGE

	Under 60 yrs		*Over 60 yrs*	
Vertebra	*(kp)*	*(lb)*	*(kp)*	*(lb)*
L1	520	1144	270	594
L2	600	1320	260	572
L3	635	1397	250	550
L4	650	1430	270	594
L5	590	1298	240	528

All analytic determinations mentioned thus far were performed in vitro. No matter how fresh such specimens are, there has been some fluid loss. Consequently, for the in vivo dynamic conditions, there is probably still another added increment of reversible compression before fracture. Finally, Perey was able to ascertain from his investigation that end-plate resistance is similar in lateral and central areas. This helps explain the lack of any particular uniformity to the area of end-plate disruption that occurred during his testing.

TABLE IV

MEDIAN BREAKING POINTS FOR 223 VERTEBRAL END-PLATES TAKEN FROM SPECIMENS OF L1 THROUGH L5

Age (*yr*)	*Median Breaking Point* (*kp/cm²*)	(*psi*)
20-30	107	1530
31-40	98	1400
41-50	76	1085
51-60	77	1100
60	43	614

Two facts gleaned from Perey's work stand out as being particularly important. The first of these is that end-plate fractures occur at lower level loading than is required to reach the proportional limit. Second, by the time a 16% compression of a vertebral body has occurred, one or both end-plates have usually failed. Recognizing the difference between proportional limit (Perey, 1957) and yield point (Ruff, 1950), one appreciates that a loading differential exists between these two types of structural failure. One can conclude that acceptance of the "yield point" as being equivalent to irreversible compressive deformity implies even greater differentials between end-plate and vertebral body breaking points than Perey ascertained with proportional limit criteria.

We can begin to appreciate not only that end-plate disruption occurs at levels appreciably below irreversible vertebral body compression, but also, (and of greater concern) that a number of spinal column end-plates incur loss of structural integrity before demonstrable fracture of the most susceptible vertebral body. When transposed to the live ejection situation this bit of knowledge takes on pertinent clinical implications.

The investigations of Ruff (1950), Perey (1957), Roaf (1960) and Yorra (1956) demonstrated that end-plate and vertebral body damage is far more apt to occur during spinal axial loading than intervertebral disc disruption. This appears to be substantiated by the infrequent reports of disc trauma contained in the ejection literature.

Based on information obtained from the above investigations we know that the vertebral end-plates and vertebral bodies are the tolerance-limiting structures of the axially accelerated, healthy spinal column under experimental laboratory conditions. Stech recently extended Ruff's original experimental data to calculations on the remaining thoracic vertebrae. Stech appreciated the relatively constant increase in percent of body weight supported as Ruff progressed from the eighth thoracic through the fifth lumbar vertebra and postulated that this constancy also exists upward from T8. Extrapolating upward in a constant 3% decrease per vertebra, Stech arrived at a 9% value for T1. The head and neck, which theoretically is all that the first thoracic vertebra supports, has been calculated as representing approximately 9% of total body weight. By making what appears to be acceptable approximations. Stech calculated both the breaking strengths of T1-T7 and the percent body weight supported by these individual vertebrae. Table V presents both Ruff's (1950) original T8-L5 data and Stech's extrapolated T1-T7 values. Having these data Stech used the concept of probability to define spinal acceleration tolerance

(injury) curves. Using this concept of relative probability of injury, one can determine levels at which the incidence should be very low and above which the extent and severity of injury will increase with acceleration. I agree with Stech's cautioning statement that the most important fact about tolerance curves and injury probability is that the levels represented are risk levels. This means that injury will occur at these levels; that we know it will occur, and that we are willing to allow its occurrence because it represents an acceptable chance. Due to the variables which he realized affected the data gathered by Ruff and Perey, Stech made certain necessary assumptions in constructing his tolerance curves. There are two reasons, however, why the curves do represent acceptable accuracy: Stech (May 1963) estimated mean breaking level values on the low side and used variances that are almost assuredly higher than the actual unknown variances. Table VI represents Ruff's individual vertebra data which Stech corrected for age, location in the spinal column, and body weight. This table also contains an estimate of the standard deviations for these vertebrae derived on the basis of the number of specimens that Ruff tested. Table VII gives these same data in pounds with the standard deviations reestimated, using the average coefficient of variation. Stech then did a similar analysis on Perey's vertebral body and vertebral end-plate breaking strength data. Tables VIII and IX present vertebral body and vertebral end-plate breaking strengths for L1 through L5 and their respective standard deviations, corrected for a 28 year old specimen.

TABLE V

RUFF AND STECH DATA

Vertebra	*Body Weight Carried (%)*	*Weight Carried 160-lb man*	*Break Strength (lb)*	*Breaking Load G*
T1	9	14.4	360	25
T2	12	19.2	480	25
T3	15	24.0	600	25
T4	18	28.8	720	25
T5	21	33.6	840	25
T6	25	40.0	1000	25
T7	29	46.4	1160	25
T8	33	52.8	1315	24.9
T9	37	59.2	1493	25.2
T10	40	64.0	1632	25.5
T11	44	70.4	1700	24.2
T12	47	75.2	1757	23.4
L1	50	80.0	1790	22.4
L2	53	84.8	1925	22.7
L3	56	89.6	2161	24.1
L4	58	92.8	2168	23.4
L5	60	96.0	2366	24.6

TABLE VI

RUFF'S DATA IN REDUCED FORM

Vertebra	*Average Strength* ($\bar{x}$ *in kg*)	*Standard Deviation* (σ *in kg*)	*Coefficient of Variation* (*V*)
T8	534	16.5	32.3
T9	618	40.8	15.1
T10	647	69.5	9.3
T11	688	49.1	14.0
T12	706	55.4	12.7
L1	721	47.7	15.1
L2	761	75.6	10.1
L3	862	70.9	12.2
L4	(855)*	—	—
L5	898	72.7	12.4

*Single data point

TABLE VII

RUFF'S DATA IN FINAL REDUCED FORM

Vertebra	*Average Strength* ($\bar{x}$ *in lb*)	*Standard Deviation* (σ *in lb*)
T8	1175	79.6
T9	1363	92.2
T10	1427	96.4
T11	1517	102.5
T12	1557	105.2
L1	1580	107.4
L2	1678	113.3
L3	1901	128.3
L4	1940	131.2
L5	1980	133.8

TABLE VIII

PEREY'S DATA FOR VERTEBRAL BODIES IN STECH'S FINAL REDUCED FORM (FOR AGE 27.9)

Vertebra	*Mean Breaking Strength* (*lb*)	*Standard Deviation* (*lb*)
L1	1266	362
L2	1383	395
L3	1395	399
L4	1415	404
L5	1661	475

TABLE IX

DISTRIBUTION OF END-PLATE BREAKING STRENGTH (STECH)

Vertebra	*Mean Breaking Strength (lb)*	*Standard Deviation (lb)*
L1	982	280
L2	1063	305
L3	1112	316
L4	1178	338
L5	1194	343

Figure 8 graphically illustrates Stech's curves on probability of damage for T8-T12 vertebrae during steady state acceleration. *Figure 9*, which presents similarly plotted curves for L1 to L5 proportional limits, compressive limits and end-plate limits, demonstrates most adequately one of

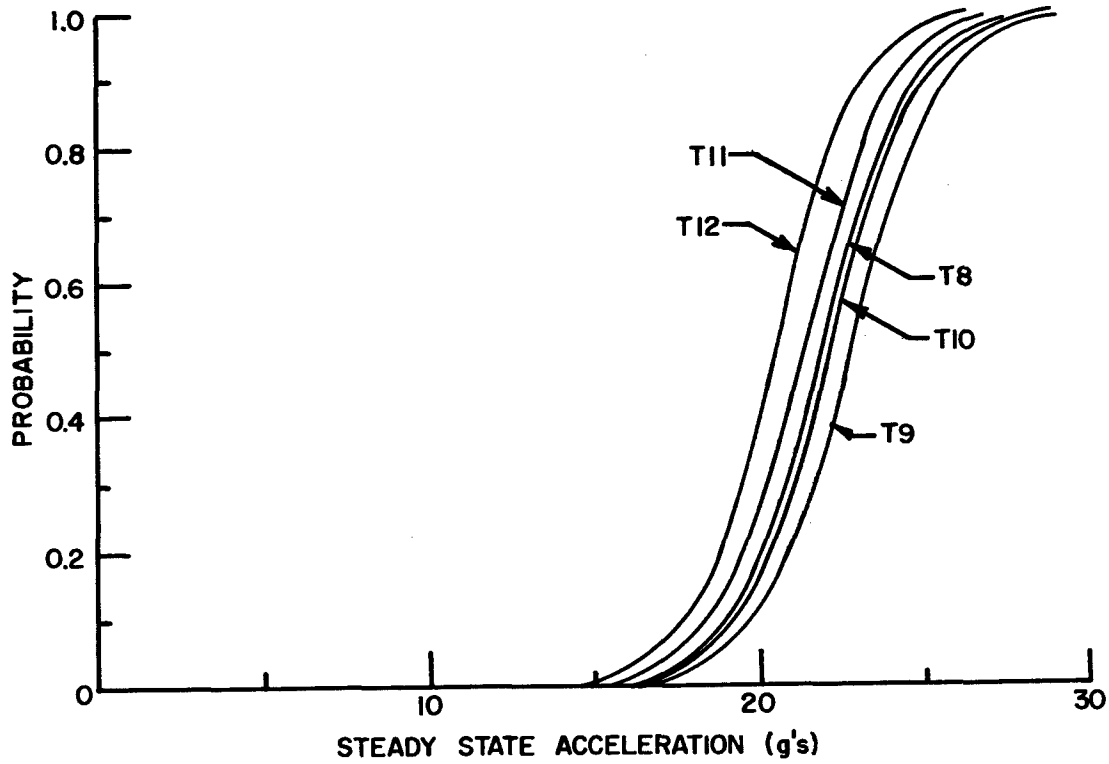


Figure 8. Stech's Curves on Probability for T8-T12 Vertebrae during Steady State Acceleration (from Stech, May 1963)

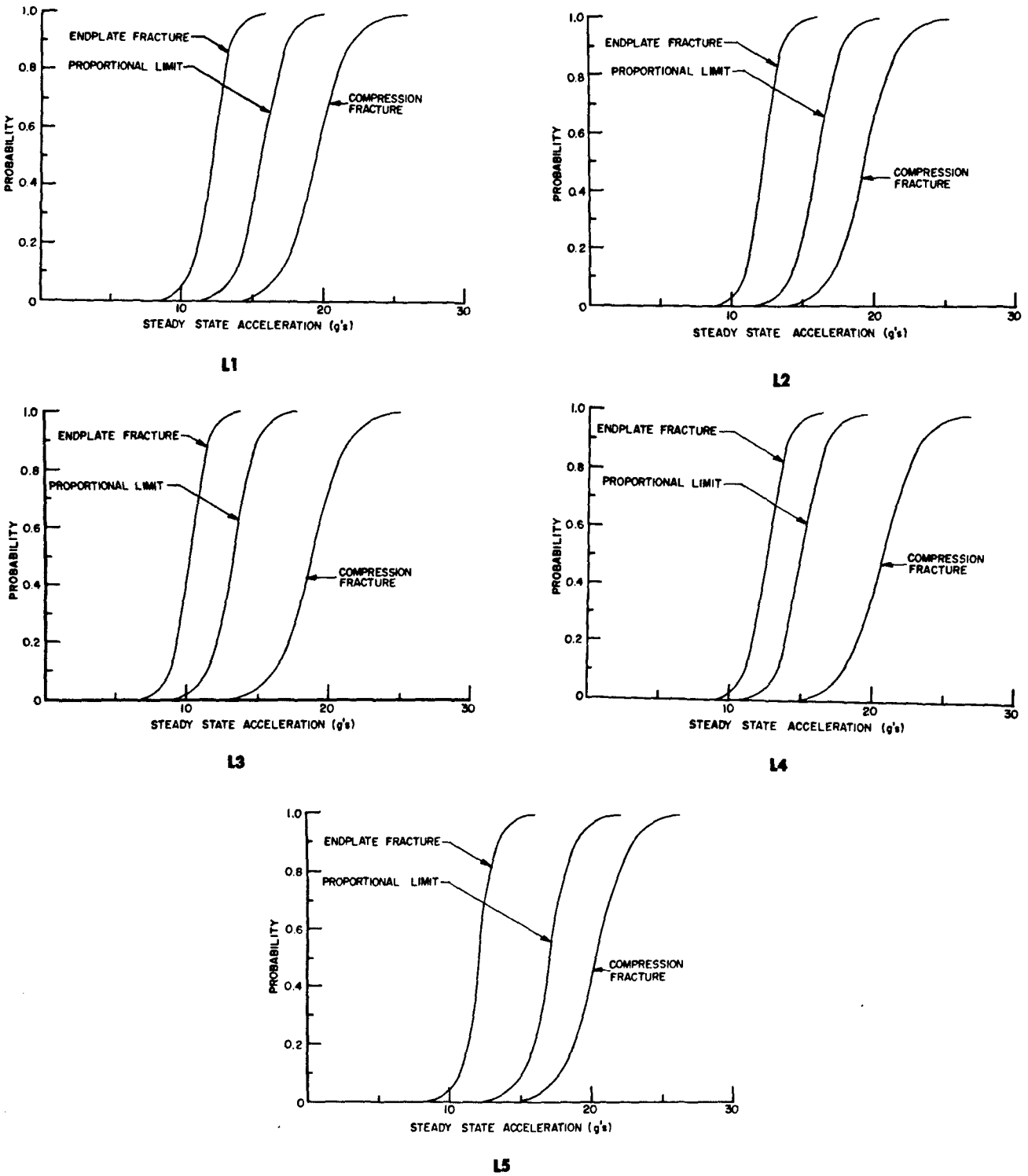


Figure 9. Probability of injury curves for L1-L5 vertebrae during steady state acceleration.

the points emphasized in this section. End-plate fractures occur at input levels below those required for compressive vertebral body fracture. Stech recognized that his curves, which are representative of one age group and a fixed acceleration, could not be transposed to the age and acceleration variable operational situation. Knowing the relationship between the age and breaking strength of vertebrae and acknowledging the acceleration profile variability of catapult performance, Stech (May 1963) plotted the curves shown in *figure 10* for both catapult steady G performance and age variabilities. In the final analysis of his data Stech wished to transpose probability of injury curves for single vertebrae to information on the entire column. This is especially pertinent in view of the following. Certain vertebrae, i.e., T12 and L1 are repeatedly fractured during ejection sequences. However, enough other vertebrae are fractured sufficiently often that the supposedly "weakest" vertebra can, all factors considered, be presumed to be also variable in position. Since this total susceptibility to injury is variable, probability of injury curves for T12 and L1 cover the majority but not all cases.

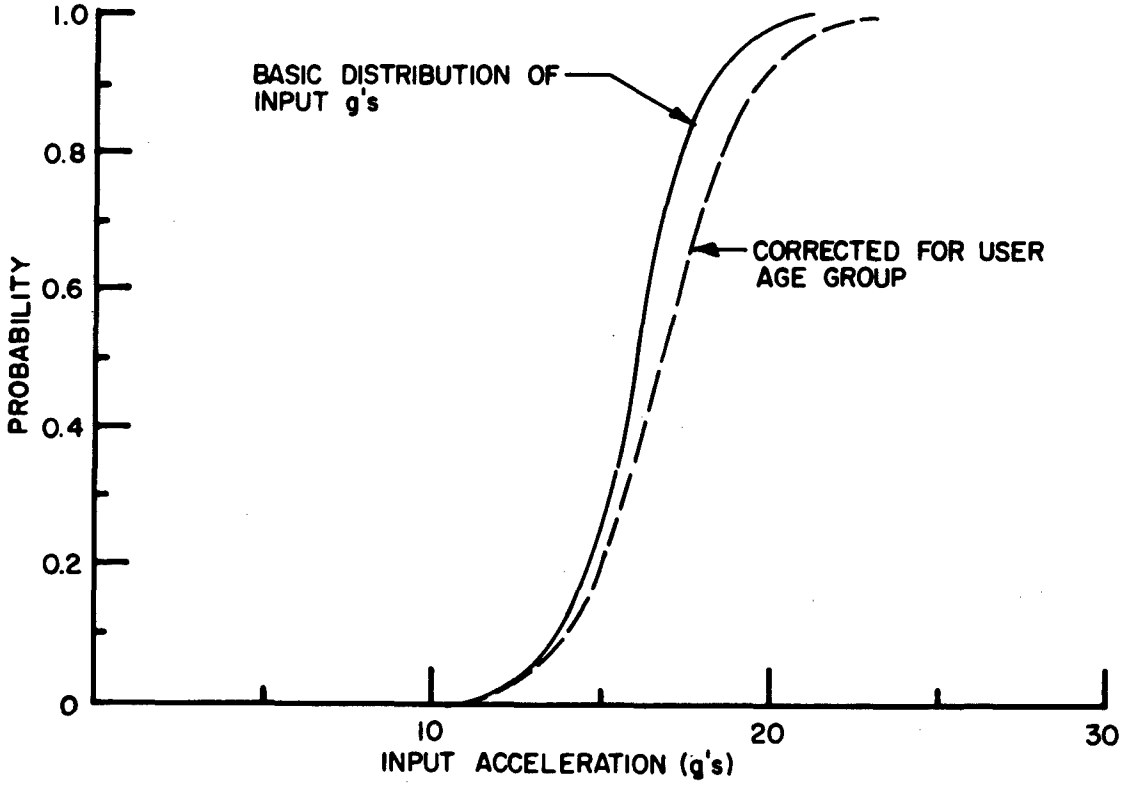


Figure 10. Distribution of Input Acceleration — Basic and Corrected

"The other side of the coin" can also be examined for the probability of no injury. Using the product of the probabilities of no injury for all 17 thoracic and lumbar vertebrae, Stech calculated the probability of injury curves for the entire dorso-lumbar column. *Figure 11* illustrates these curves for live spinal columns at ages 20, 25, 30, 35 and 40.

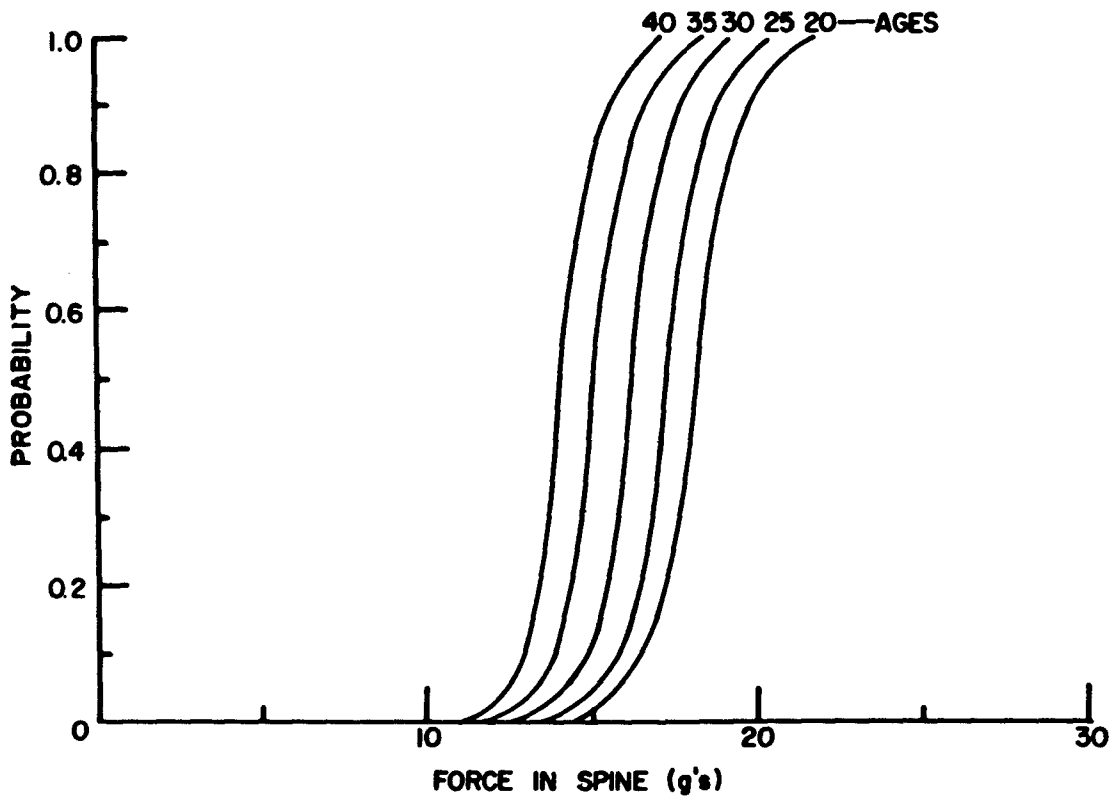


Figure 11. Stech's Probability of Injury Curves for the Dorso-Lumbar Column for Various Age Groups

As was mentioned before describing these probability-of-injury curves, Stech estimated mean values on the low side and used variances that are probably higher than true variances. Stech's curves most likely indicate a higher probability of vertebral body fracture at a given G level than is in actuality true. The curves should be used cautiously for probabilities below 0.1 or above 0.9 and the age distribution of the potential ejectee population should be used to generate the operational curves for those groups. Finally, the acceleration-time history of an ejection profile does have a significant effect on an operational probability of injury curve and must therefore be specified or known when using such curves.

SPINAL STRUCTURE UNDER DYNAMIC MECHANICAL LOADING

During the past 25 years of ejection system design, acceleration levels that have been suggested and used were in part empirically established with the aid of experimental laboratory data and validated largely through trial and error. More recently, considerable precision has been gained through dynamic analog models. Before being appalled at, and hypercritical of what may appear to be gross design criteria, the reader should recall that such systems had to be rapidly developed for life-compromising situations.

In reviewing these suggested tolerance levels I will also review the relationship between a particular acceleration profile and instances of spinal fracture or pain occurring within that pro-

file. Pain is mentioned, at this point, to emphasize one of the reasons for which this report was written. Persistent pain, in the presence of normal x-rays, may well be indicative of hidden endplate fracture. Section VI fully explores the more elaborate diagnostic aids available to document such an injury which may be missed on routinely obtained roentgenograms.

In attempting to delineate acceptable ejection tolerance levels, the Germans in 1941-42, following Ruff's work (1950) and their own ejection tower trials, believed that 18 G could be sustained without harm. Early German experience did, however, yield compressive fractures at levels below 15 G. This was before they were aware of the importance of restraint harness, seat cushion and the rate of acceleration onset. Wiesehofer (1943) cautioned that spinal trauma was a function of not only the peak G attained during the profile but also the rate of onset associated with catapult firing. That the Germans did not incur more fractures is surprising because some of their early systems, which only peaked to 12 G, did so in the first 10 msec of the profile! By 1944-45 German ejection velocities had substantially increased above earlier 8-9 meters/sec. In addition they were tolerating 18-20 G peaks with much less spinal trauma. The primary reason for both of the aforementioned was because the slower onset systems reached peak level later in the acceleration profile.

Swedish catapults, designed between 1944 and 1947, had a top acceleration of approximately 21 G. Their seat acceleration reached this level after about 70 msec during which the onset rate did not exceed 300 G/sec. The peak velocity of these systems was about 17 meters/sec.

British experience began with the Martin-Baker investigations. In early 1945, a compression fracture was incurred at a level below 12 G. Investigation revealed that the onset rates of the early British ejection tower exposures were frequently 600-800 G/sec. As a result of further exposures at substantially lower onset rates the British, in late 1945, accepted the following parameters for catapult design: peak acceleration should not exceed 21 G and this peak should not be held longer than 100 msec; rate of onset of acceleration should not exceed 250-300 G/sec.

U. S. ejection catapult specifications necessary to optimize chances of safe escape from aircraft were accurately defined between 1945 and 1947. Ames (1947-1948), aware of the increased dynamic overshooting that occurs with high onset rates, cautioned during that period that such overshoot would be negligible with onset rates below 200 G/sec. In 1947, ejection seat equipment developed by the Army Air Forces and the Ordnance Department provided a terminal velocity of 60 ft/sec with a maximum of 14-16 G on the subject at a rate of 175 to 200 G/sec.

Although the Germans were aware of the upper torso support that resulted from use of armrests, and the British noted the prevention of upper body flexion afforded by face curtains, it was Ames (1947) and Watts (1947) who further investigated the exact role played by both of these. Ames, in 1947, calculated that properly used armrests would relieve as much as 30 to 50% of the load carried by the lumbar vertebrae. Also in 1947, Watts, in comparing armrest with face curtain ejection tower exposures, noted more complaints at substantially lower levels (10-14 G) from individuals using armrests. When these same subjects used face curtains they repeatedly tolerated 17-21 G levels without significant complaint. Although Watts documented three instances of vertebral fracture at 16-19 G levels he concluded that ejection seats designed to peak at 18-21 G should be tolerated without injury by the majority of the pilot population. Ames advised 20 G upper limit as did Glasser (1950). In 1955 Martin stated that both the 60 ft/sec and 83 ft/sec Martin-Baker seats had onset rates of 200-250 G/sec and peak levels of 18-21 G. Barash (1956) empirically stated that tolerance limit is about 20 G for 100 m/sec or 25-28 G for 10 m/sec.

Results from ejection systems designed according to these acceleration profile specifications have been gathered slowly. From 1950 to the present, a number of reports, documenting injuries

received utilizing seats with known accelerative profiles, have appeared in the aerospace literature. Three of the most enlightening of these are reports by Laurel and Nachemson (1963), Fryer (1961), and Jones et al (1964). Laurel and Nachemson described 23 ejection profiles containing 15-20 G peaks without a single incidence of fracture, but noted 12 incidents of fracture in 29 ejections containing 20-25 G profiles.

Fryer (1961) described British experience with the Martin-Baker seat (18-21 G peak) from 1949 to 1960. There were 41 cases of x-ray proven fracture out of 220 ejections (19%). In this series of 41 there was an average of two fractures per spinal column. *Figure 12* reveals the particular susceptibility of T12 and L1 which were fractured 18 and 10 times, respectively. Fryer noted that 27 of the 41 fracture cases occurred with the 80 ft/sec catapult. Therefore, based on the reports by Laurel and Nachemson (1963) and Fryer, a 20% increase in peak G and a 25% increase in terminal velocity resulted in a significantly increased incidence of spinal fracture.

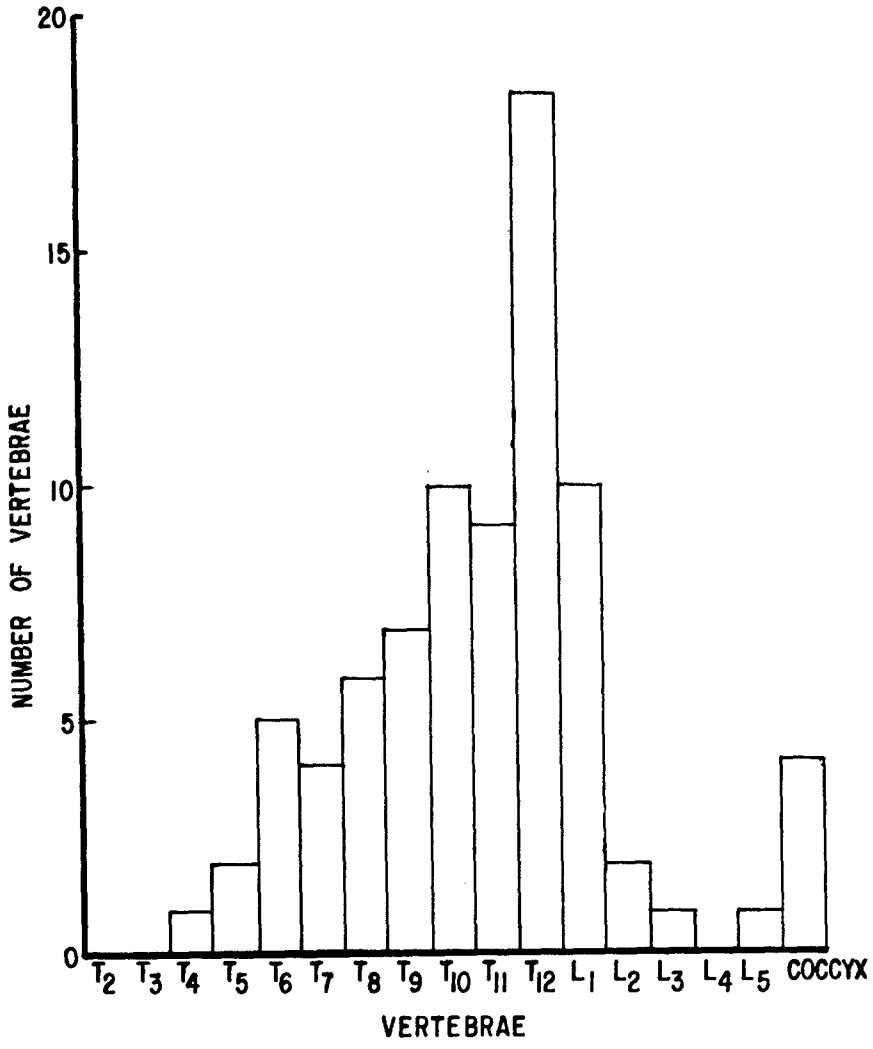


Figure 12. Incidence of Vertebral Injury in Aircrew Surviving Ejection (from Fryer, 1961)

Jones et al (1964) documented the 1958-1963 incidence of spinal fracture from the 18-21 G Martin-Baker seat used by Britain, the United States, and Sweden during that period. Frequency of fracture was comparable for the British and U. S. being 19 and 21%, respectively. The Swedish incidence, however, was 46%. *Figure 13* from Jones data substantiates Fryer's experience of T12 and L1 fracture preponderance. The most susceptible vertebra again appears to be T12, which compresses 1.5 times as often as the next most frequently fractured T8, T9, and T10. The preponderance of ejection-incurred vertebral fractures lies in the T12, L1 segment of the spinal column. It has been felt in the past, and presently is not an uncommon belief, that ejection fractures tend to occur in a higher region of the column than do other accidental compressions.

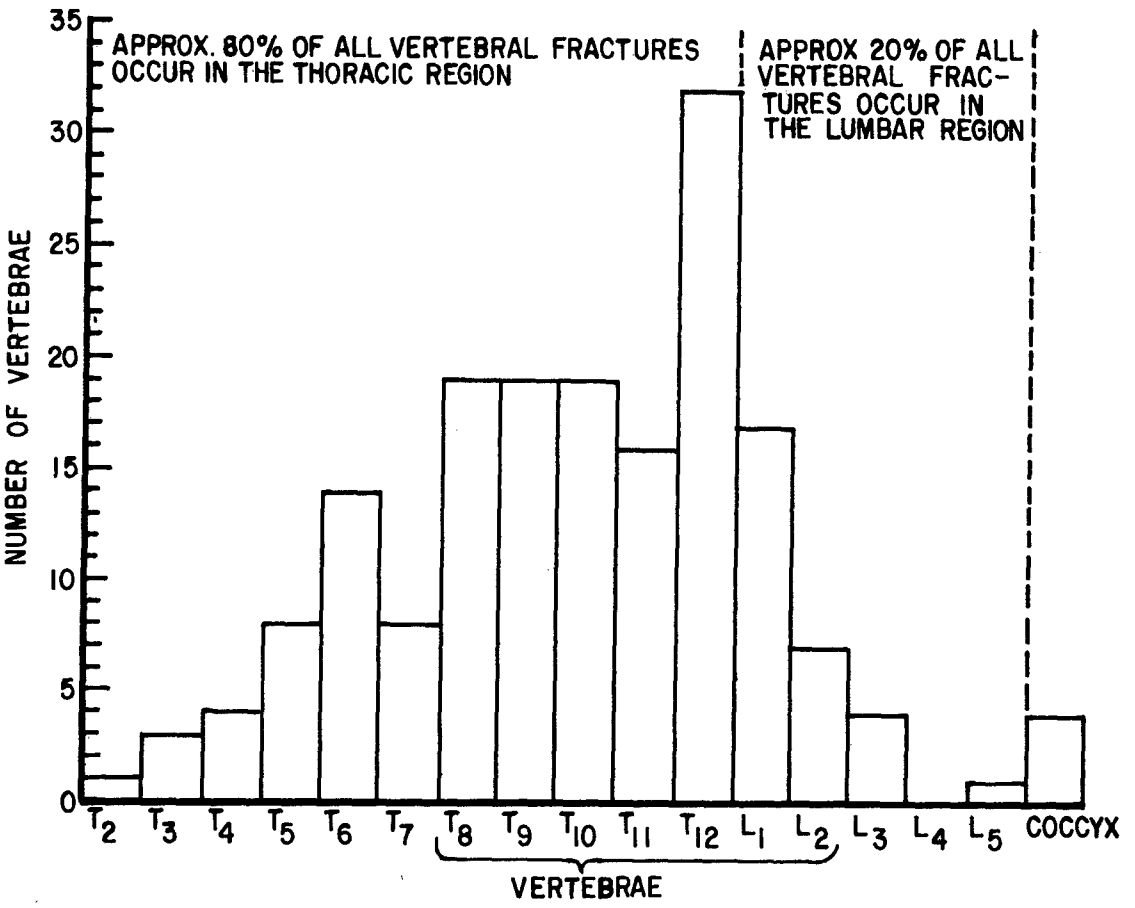


Figure 13. Incidence of Vertebral Injury in Aircrew Surviving Ejection (Jones et al, 1964)

Jefferson's data (1928), however, as revealed by *figure 14*, appears to partially refute this belief. Examination of the graph reveals a spike in the T12-L1 region illustrating similar force concentration on a few particular vertebral segments during the trauma of nonejection type injuries. The high incidence of fracture in this particular area, through which physiologic flexion occurs, appears to confirm the orthopedic postulate that a "spinal column tends to flex during compression."

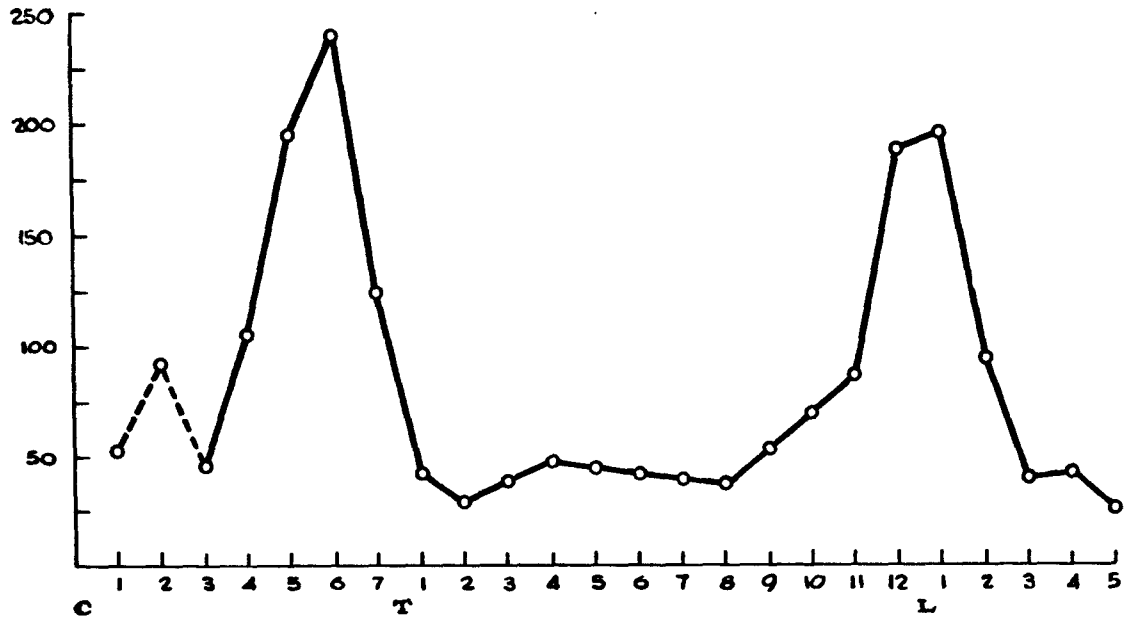


Figure 14. Localization of Spinal Fracture in 2006 Cases (from Jefferson, 1928)

The acceleration profile of most present day upward catapult seats averages 200-300 G/sec onset, 22 G max, 70 ± 10 ft/sec terminal velocity and 10-80 milliseconds of peak G exposure. Depending upon the rate of onset that may initiate a dynamic response within the individual, these levels could be slightly higher as actually experienced. Using such an acceleration profile under operational conditions results in an incidence of spinal column compression that averages between 19 and 25%. For each incidence of spinal injury there is an average of two vertebrae fractured. In Fryer's series (1961), 47 (59%) of 80 individual fractured vertebrae were in the T10-L1 region. In Jones and associates (1964) series of 178 fractured vertebral bodies, 84 (47%) were in the T10-L1 region. Since the two lowest "minimum G" breaking forces calculated by Ruff (1950) from his test data were for T12 and L1 (18.6 G and 18.2 G, respectively), one gains some respect for the potential accuracy of transposing carefully acquired cadaveric data to the living body. These reports reveal only the incidents of demonstrable decreased vertebral height. Fryer did record 28 incidences of minor injury, which he defined as painful spinal symptoms or signs in the presence of normal x-rays. At present there is no way of knowing how many end-plate fractures (some painful and others asymptomatic) occurred during these ejections.

Little is known at present about the possible long term sequelae of undetected end-plate fracture. It is significant, as Jones (1964) notes, that of the first seven crewmen who incurred vertebral fractures in Martin-Baker seat ejections, five were retired because of one of the following complications: radiculoneuritis, degeneration of intervertebral disc with localized arthritis, and arthritis with muscle spasm. One cannot help but wonder how many undetected end-plate fractures, with or without later compression, will result in similar future difficulties. Even though acceleration profiles are designed above end-plate breaking limits, end-plate fracture in itself is a structural tolerance endpoint. At the present time this is not sufficiently understood by engineers and not often considered or diagnosed by medical personnel.

Use of dynamic analog models to evaluate the spinal injury potential of ejection forces has provided the ejection seat designer with a considerably more accurate measure of "human tolerance limits." In appraising spinal stresses occuring during ejection acceleration it is important to realize that the spine may receive forces greater than those received by the seat. By virtue of the structural composition of the human body, the vertebral column is part of an elastic system capable of a dynamic response. The elasticity arises out of the flexion, compression, and expansion properties of biologic tissues. Being part of an elastic system, and being in itself elastic; the column in connection with the body masses coupled to it responds to high-onset accelerative forces transmitted to its caudal end by compression and bending, and by subsequent expansion. Depending upon the rate of onset of acceleration during the initial phase of ejection, the upper end of the spine may not begin to move upward until the inelastic seat pan has already begun dynamic spinal compression. Up to this stage, under such conditions, the seat is moving faster than the upper end of the spine, which afterwards, in order to catch up, must exceed seat acceleration. In surpassing seat acceleration, the spine receives additional force loading in overcoming the inertia of the upper torso. The resulting dynamic response or overshoot of the spine can result in accelerations on parts of the subject that are higher than those on the seat. In particular, this can be of importance, as has been observed and reported by many investigators, if an elastic seat cushion is placed between subject and seat pan.

Since Latham's early work (1957) on the dynamic response function of seated human subjects, considerable progress has been made in the measurement, interpretation and analytical expression of this dynamic response. Today the injury potential of complex acceleration time functions, untested with respect to their biological effectiveness, is probably best evaluated by means of these analytical methods and the model concepts on which they are based. Special analog computers are available to calculate the dynamic response of the seated subjects when using different types of seat cushions or restraints. It is not the purpose of this discussion to review critically this area of impact research. Its main result is an index of equal spinal injury potential which is consistent with higher acceleration inputs for shorter impact durations similar to Ruff's graph in *figure 7*. The injury potential of acceleration-time patterns with long rise times is less (in a quantitatively predictable way) than the injury potential of patterns with short rise times and equal peak acceleration. It can be shown that the injury probability curves for static loading discussed in previous sections combined with the present knowledge of body dynamics permit derivation of injury probability curves for various acceleration-time patterns produced by ejection systems.

When human exposure to any environmental stress is necessary, well-defined tolerance limits for the most susceptible organ system are desirable in design of that environment. Because of the multiple variables encountered in both the human body and the acceleration environment, precise definition of these limits are not always feasible. The ideal solution would be to outline ejection tolerance curves that will be safe for the average ejectee population. The word "safe,"

however, requires qualification. Any tolerance levels defined for maintaining functional spinal column integrity will, for the most part, be above the breaking strength levels of the weakest link in the column, i.e., the vertebral end-plate. Since ejection constitutes a life-saving situation without alternatives, and end-plate fracture does not usually result in functional disability, such vertebral body tolerance curves are acceptable. Presently accepted and used seat-to-head spinal accelerative profiles should be maintained as roughly representative of spinal tolerance for the young, healthy, properly positioned and adequately restrained vertebral column. Examination of the injury curves generated by Stech (May 1963) will give an acceptable fracture-risk figure for a particular set of human-biodynamic conditions within a particular ejection acceleration profile. However, as evidenced by the sequelae of the first five fractures resulting from Martin-Baker seat ejections, spinal trauma is not always a benign, self-limited affliction. The long term effects of even minor bony spinal injury have not been sufficiently described to give an accurate idea of the possible future sequelae of such injury. All individuals who in any way deal with ejection seat design or testing, as well as physicians who come in contact with postejection back complaints, should be aware of and have an appreciation for the above mentioned characteristics of spinal biodynamics. With present day knowledge, materials and instrumentation capabilities, intelligent man is able to design, construct and operate systems that will repeatedly function within predetermined specifications. A particular ejection system should be capable of being used by a majority of specifically chosen individuals, in most instances, and under specified conditions, such that a predictable spinal injury rate will prevail. The fractures that do occur should result because of variation in those parameters outside of objective control, such as the congenitally abnormal vertebra and the hyperdynamically responsive spine.

SECTION V.

Pathogenesis of Ejection Spinal Fracture

In the young, physically sound, properly trained, pilot population, the incidence of spinal fracture occurring during ejections with tested, reliable systems should be minimal. However, in addition to the biologic variability wherein predisposed vertebrae may fail at acceleration levels below the normal structural tolerance, there are a number of associated factors that contribute to the difference between an intact or damaged spinal column after ejection.

The acceleration arising from the catapult or rocket firing of an ejection seat is dependent upon the performance variability of the propellant or propellant mixtures and the prevailing environment at the time of propellant ignition. Present day systems, however, have usually been qualified within high confidence-reliability specifications, which optimize chances for predictable acceleration profiles during most operational conditions. Other variables influencing an ejection acceleration profile are the individual's body somatotype and dynamic center of gravity as well as the airspeed and orientation of a failing aircraft. Added to these variables are the factors of restraint harness slack or preload, and the interrelationship between seat cushion elasticity and dynamic response.

Pilot age also influences the incidence of injury and, indeed, Fryer (1961) has noted that pilots over age 25 incur more fractures than their 19 to 24 year old counterparts. Appreciating the complex subjective and objective factors influencing the ejection sequence, one can anticipate that some benefit should be gained from ejection tower training. Farmer's analysis (1962), which disclosed a lower incidence of ejection spinal injury in individuals who had had preflight tower exposures, confirms this. However, training tower exposures have produced a significant percent of an injury which is for the most part limited to this particular environment. This is pelvic coccygeal fracture which is rare in the operational situation. This has been extensively investigated by Cooper (1963).

At the United States Air Force School of Aerospace Medicine in 1960, 200 training firings resulted in a total of four coccygeal fractures, two coccygeal separations and five "minor but discrete" coccygeal injuries. These were incurred on an MH-15 seat designed to give a peak 9 G at a rate of onset around 100 G/sec. In order to assess more precisely what was occurring, Cooper's group investigated 100 training tower ejections during which both the man and the seat were instrumented. They observed that the maximum seat accelerations were considerably higher than mean seat accelerations. Also, and as alluded to in previous sections, the higher acceleration recorded on the man indicated significant dynamic overshoot. Table X, taken from Cooper's data (1963) reveals the differences between design specifications and the actual performance of the seat catapult. The reason for such coccygeal injury is probably insufficient buttock-seat contact. Under such instances a relatively small area, the coccyx, selectively absorbs a force profile that should be distributed over the entire buttock and upper thighs. The fact that no vertebral injury occurred is not surprising since the acceleration levels experienced were below vertebral body structural tolerance limits. Although not previously mentioned in this report, most workers are aware of the necessity for adequate buttock-seat surface area contact, if coccygeal injury is to be prevented and the ejected column above is to be adequately stabilized.

The majority of factors contributing to compressive ejection fracture are vulnerable to both subjective and objective control. Thus, subject posture and restraint should be specified within rigid limits. As alluded to earlier, human spinal posture has been teleologically designed in such

TABLE X

COOPER'S EJECTION TRAINING TOWER DATA (1963)

Design Specifications		*Results*	
		Seat	*Man on Seat*
Maximum Velocity	32 ft/sec	41 ft/sec	41 ft/sec
Maximum Acceleration	9.0 G	9.4 G	13.7 G
Maximum Onset Rate	100 G/sec	250 G/sec	525 G/sec
Maximum Ejection Height	15 ft	17 ft, 9 in.	17 ft, 9 in.

manner that maintenance of the four normal spinal curves during weight-bearing and axial accelerative loading results in symmetrical load distribution and enhanced safe loading capability. The importance of this simple, biomechanical fact cannot be overemphasized when considering seat-to-head spinal stress in which unsymmetrical vertebra-disc load distribution and transmission may cause early structural failure. The inherent dangers of dorsolumbar anterior lip overloading during flexion and posterior lip overloading during hyperextension have been recognized but not thoroughly appreciated. During the initial phase of ejection acceleration there is a forceful tendency for cervical and dorsolumbar flexion to the degree that an individual who is unrestrained or poorly restrained will experience spinal bending. If such spinal flexion is sufficient, the major portion of an acceleration force vector may be concentrated along the anterior superior and inferior margins of a few vertebrae *(figure 15)*. Inadvertant pilot slumping, grasp of poorly positioned D-rings and inadequately designed arm rests, which require even minimal spinal flexion for their use, are significant contributing factors towards ejection spinal fracture. Vigorous pull on a D-ring may, besides contributing to spinal flexion and anterior vertebral lip overloading, causes preloading as illustrated in *figure 16*.

In our laboratory we recently encountered an interesting and pertinent incident of spinal fracture resulting from drop tower impact. In this incident the deceleration profile experienced was significantly below accepted vertebral structural tolerance. Review of high-speed movies revealed that the subject pulling on his thigh restraints (which simulated an operational D-ring), probably caused spinal preloading and contributed to compressive fracture of T4 and T5 vertebrae. This mechanism of spinal preloading, as illustrated in *figure 20*, can result from ejection initiation using poorly positioned D-rings, which detract from the protective capability of a well-adjusted shoulder harness system.

Seat arm rests designed for and used by a specific anthropomorphic group will partially unload the spinal axis during acceleration. Such arm supports, particularly if they contain stabilizing elbow pockets, will also tend to prevent flexion *(figure 17)*. In addition, properly used supports will contribute to the two important dynamic factors of ejection, normal postural vertebral alignment and assurance of an accelerative vector which passes directly parallel to this alignment. However, any subjective gripping of such arm rests is not isolated to hand and forearm muscular contraction. During the stressful moments of ejection initiation, hand, arm, and shoulder action are probably remarkably interrelated. When such muscular strain results in a downward pull on the shoulder girdle, as schematically illustrated in *figure 18*, it is not difficult to appreciate the degree of vertebral loading that may occur.

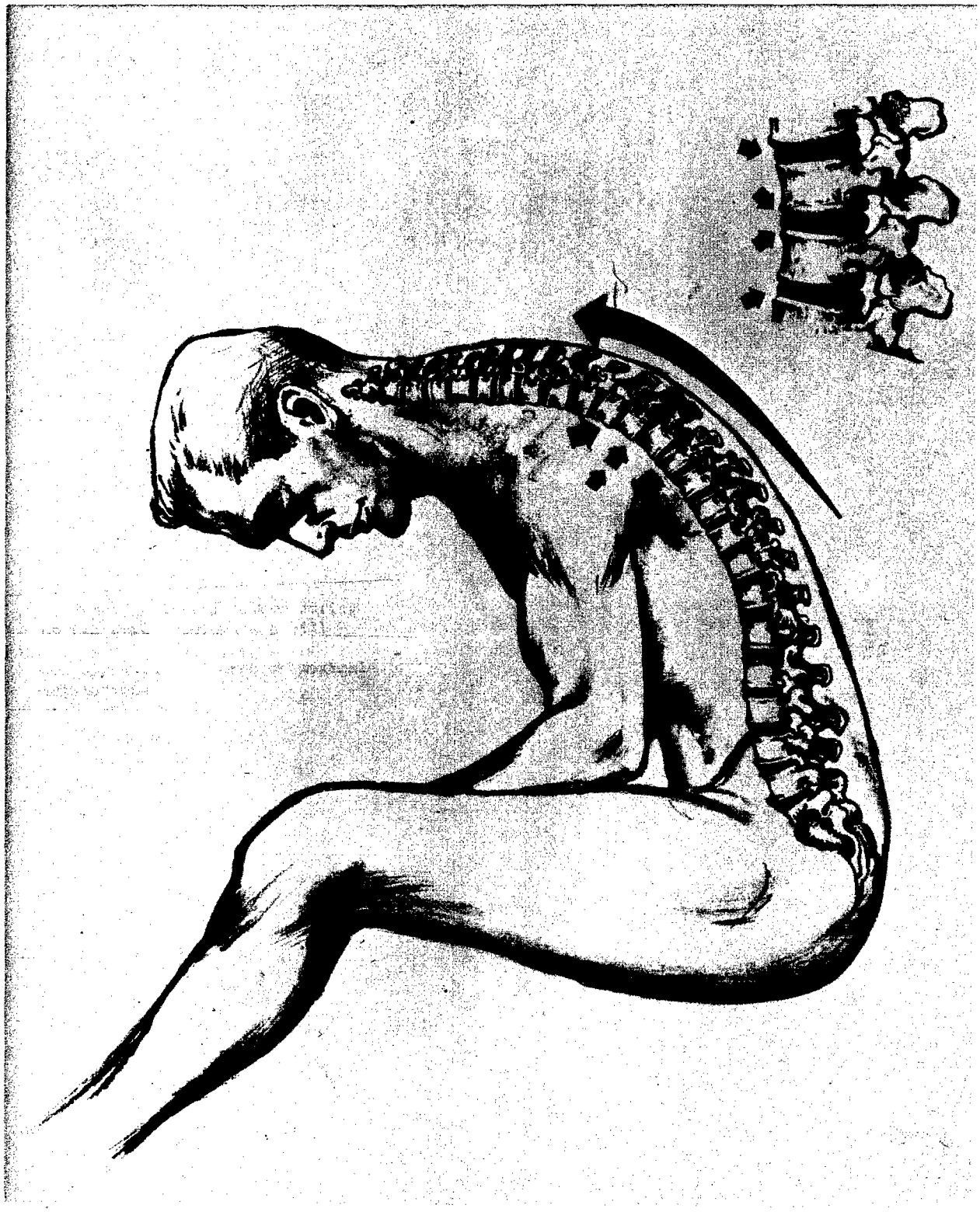


Figure 15. A flexed vertebral column during spinal axis acceleration will result in preferential anterior lip strain at the area of maximal angulation.

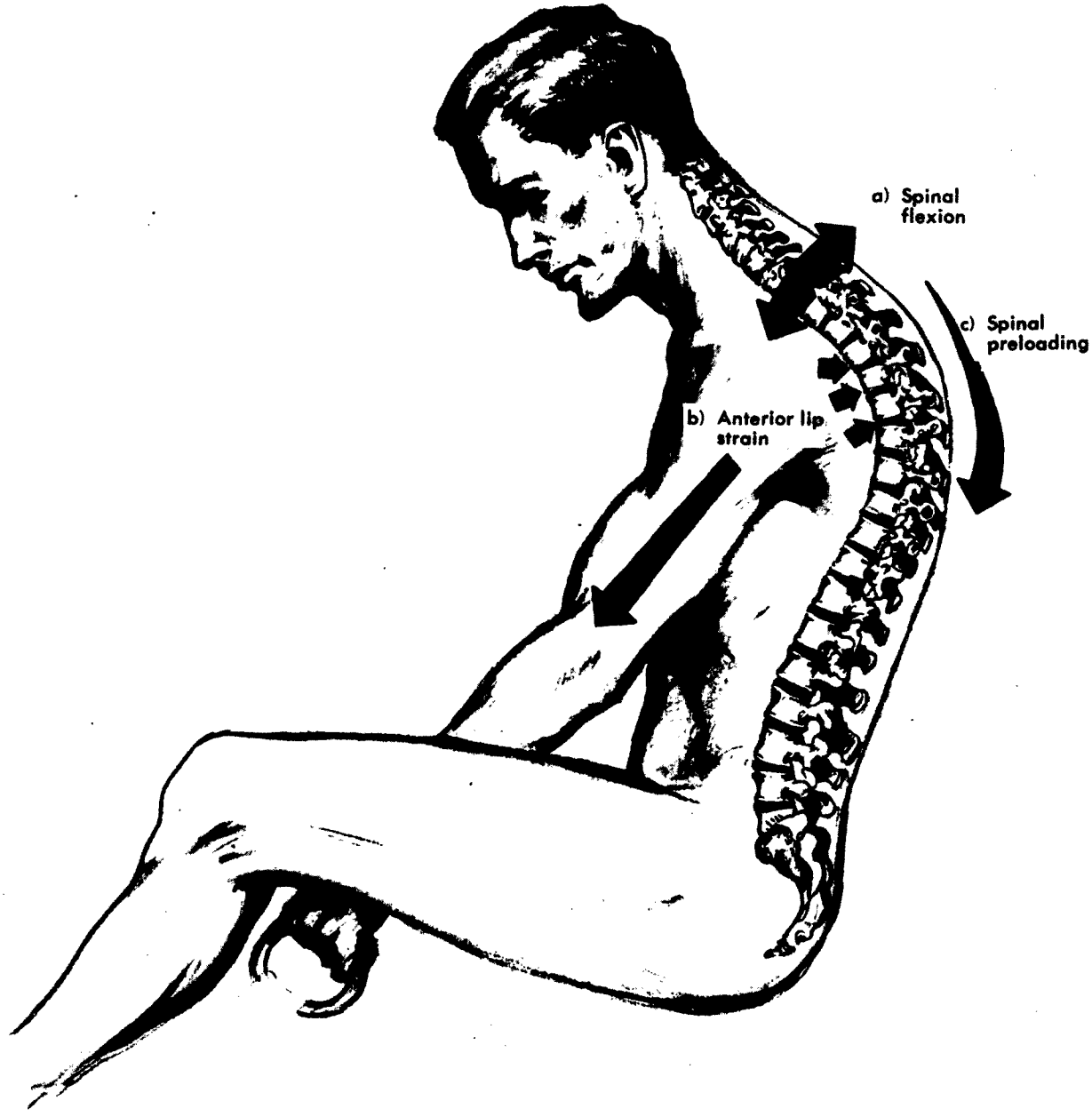


Figure 16. Muscular Strain produces (a) spinal flexion, (b) anterior strain and, (c) spinal preloading.

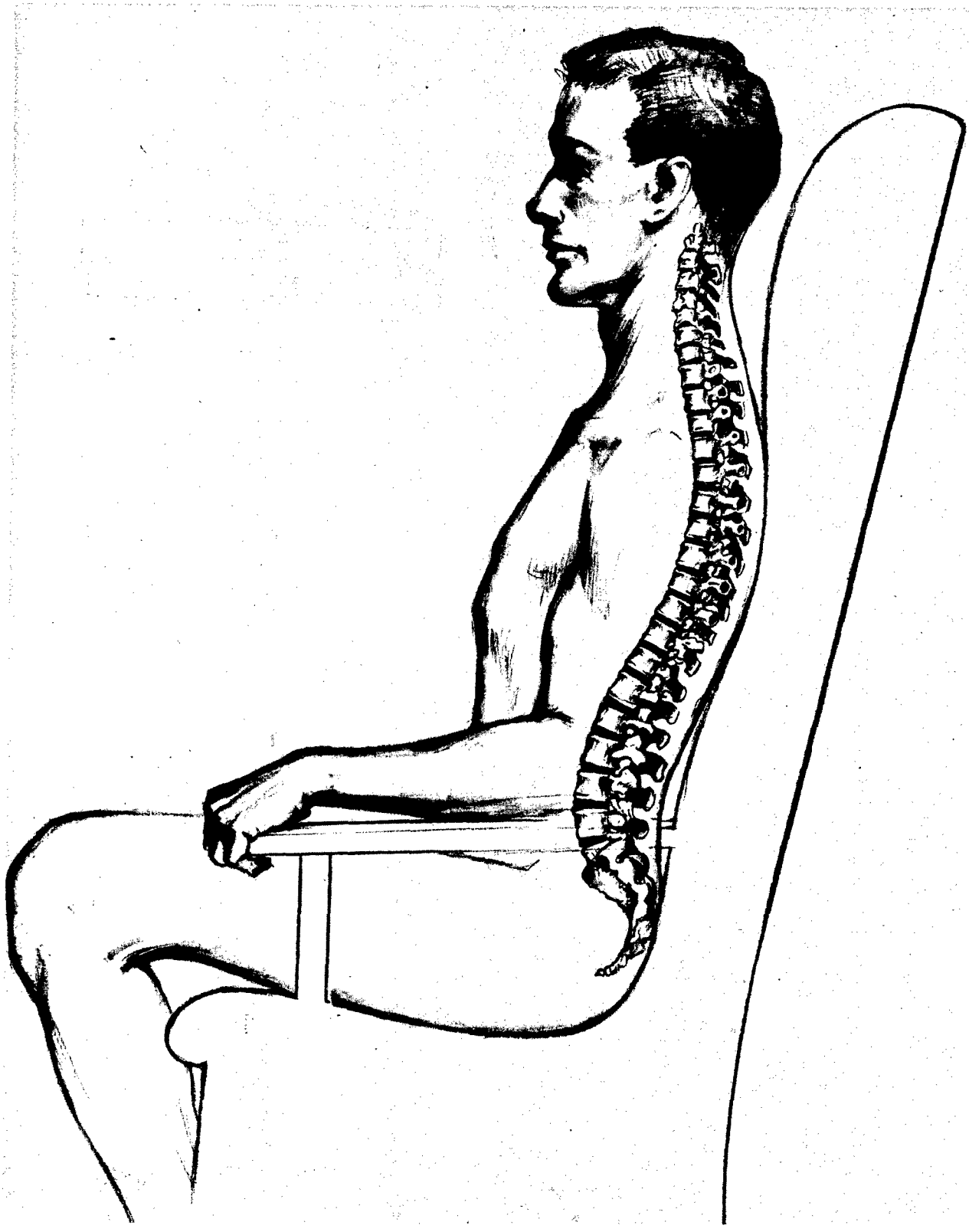


Figure 17. Arm Rests tend to maintain normal posture if the chairback end of the arm rest is high enough to support the relaxed elbows.

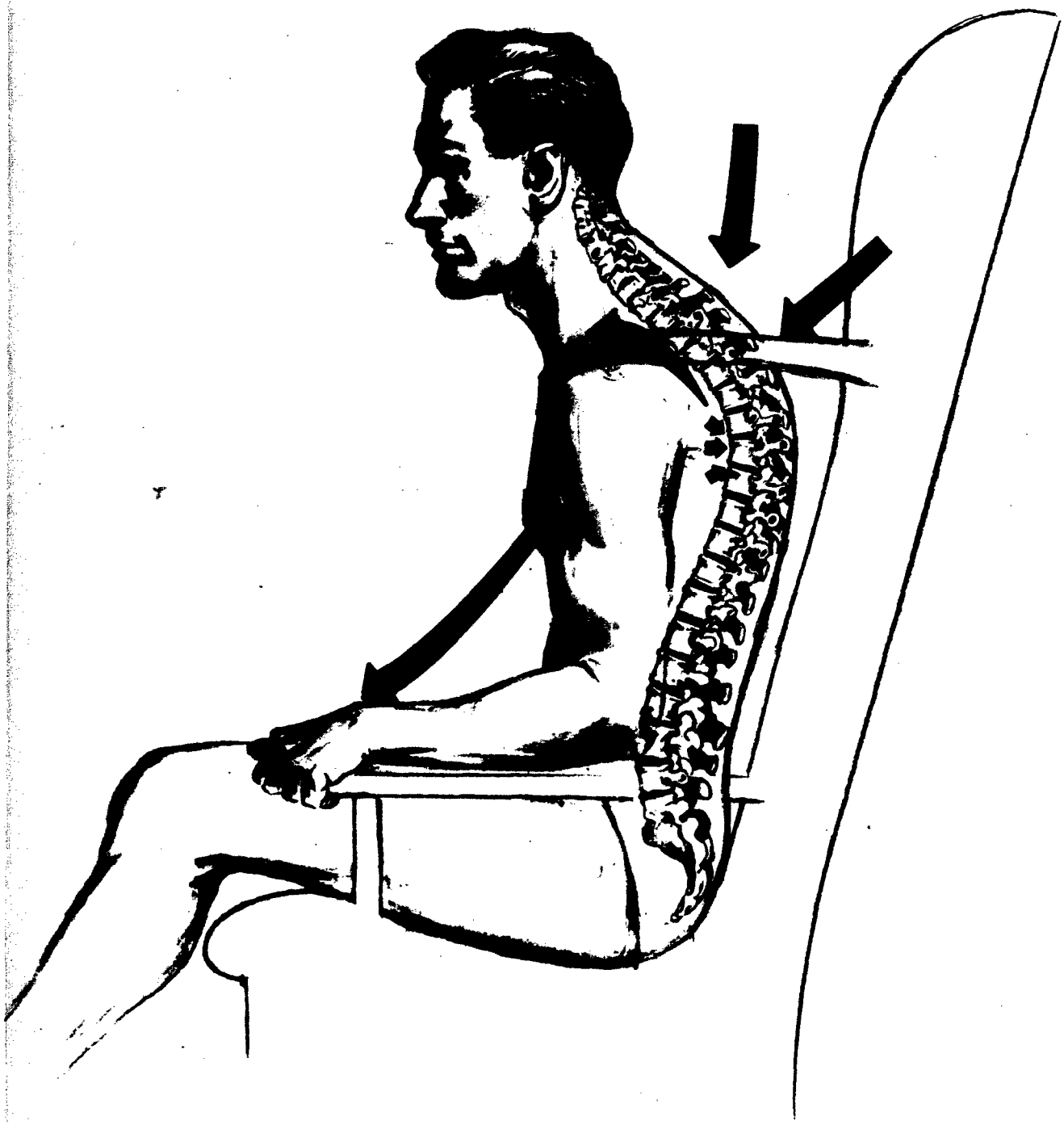


Figure 18. If strenuous muscular grasping of arm rests occurs during ejec, tion, vertebral body "overload," spinal flexion and anterior lip strain may all contribute to the main acceleration compressive loading.

In addition to poorly designed or incorrectly used arm rests and D-rings there are two other posture-restraint factors which influence injury. The first of these, the shoulder harness, should ideally both restrain the subject against the back of the seat and aid in maintaining normal thoracic kyphosis during ejection. However, an individual with a high percentile sitting height or one seated on a thick seat pack will experience a downward pull from the shoulder straps which results in vertebral preloading as illustrated in *figures 19* and *20*. An injury which in all probability arose from this mechanism has been described. The second factor concerns the use of a face curtain or overhead mode of initiating ejection. As mentioned previously, Watts, as early as 1947, stated "when exposing personnel to acceleration believed to be approaching critical levels, any method for decreasing stress on the vertebral column is advantageous." He made this statement concerning face curtain ejection initiation after noting fewer subjective complaints at higher acceleration levels during tower training than were expressed during exposures in which arm rests were used. Individuals using a face curtain type assembly repeatedly tolerated G levels in the 16-20 range without worrisome complaints, whereas these same individuals noted back pain at about 12 G when arm rests were used. High-speed filming of both types of exposures revealed markedly less upper body flexion with the face curtain.

The three advantages that properly anchored face curtains appear to have over arm rests are as follows. First, preejection flexion is impossible since the arm-shoulder positioning necessary for this mode of ejection initiation tends to maintain the normal thoracic kyphotic curve. Second, flexion during acceleration is lessened, which decreases vertebral anterior lip loading. Third, not only is the spinal axis optimally postured for safe load distribution, but the subject tends toward vertebral unloading through his overhead muscular activity. *Figure 21* schematically illustrates the protective mechanisms resulting from use of overhead ejection initiating mechanisms. These advantages, however, are predicated upon proper use and design of the face curtain. *Figure 22* depicts an unacceptable initiation technique that may contribute to spinal flexion with its potential consequences. Moreover, the speed and orientation of a failing aircraft may generate G forces that preclude the ability to reach overhead. One possible solution to this particular problem is to have the ejection system equipped with both overhead curtain and D-ring firing mechanisms. At present there is at least one aircraft being equipped with such a system.

The seat cushion is yet another factor contributing to compressive vertebral damage. An improperly designed cushion contributes to the overshoot response of the human body during acceleration. The safest cushion is in many respects direct buttocks contact with the ejection seat. This would most closely approach the ideal situation in which all points on the seat and subject could accelerate uniformly.

One last factor contributing to ejection vertebral injury is included solely for completeness is the cockpit canopy. Until a more reliable method for canopy jettison is developed or encapsulated seats are used exclusively, the degree and severity of vertebral injury occurring with through-the-canopy ejection will be related to the velocity the head achieves when it strikes the canopy. If, of course, the crewmember's head is positioned sufficiently below a "canopy striker" which adequately performs its task, there should be no significant contact between head and canopy.

Appreciation of the above factors is necessary in the design and testing of ejection systems. Exacting anthropomorphic design; adequate ejection tower training experience; properly fitted, positioned, and tightened restraint systems; proper seat cushion use; and correct spinal posture

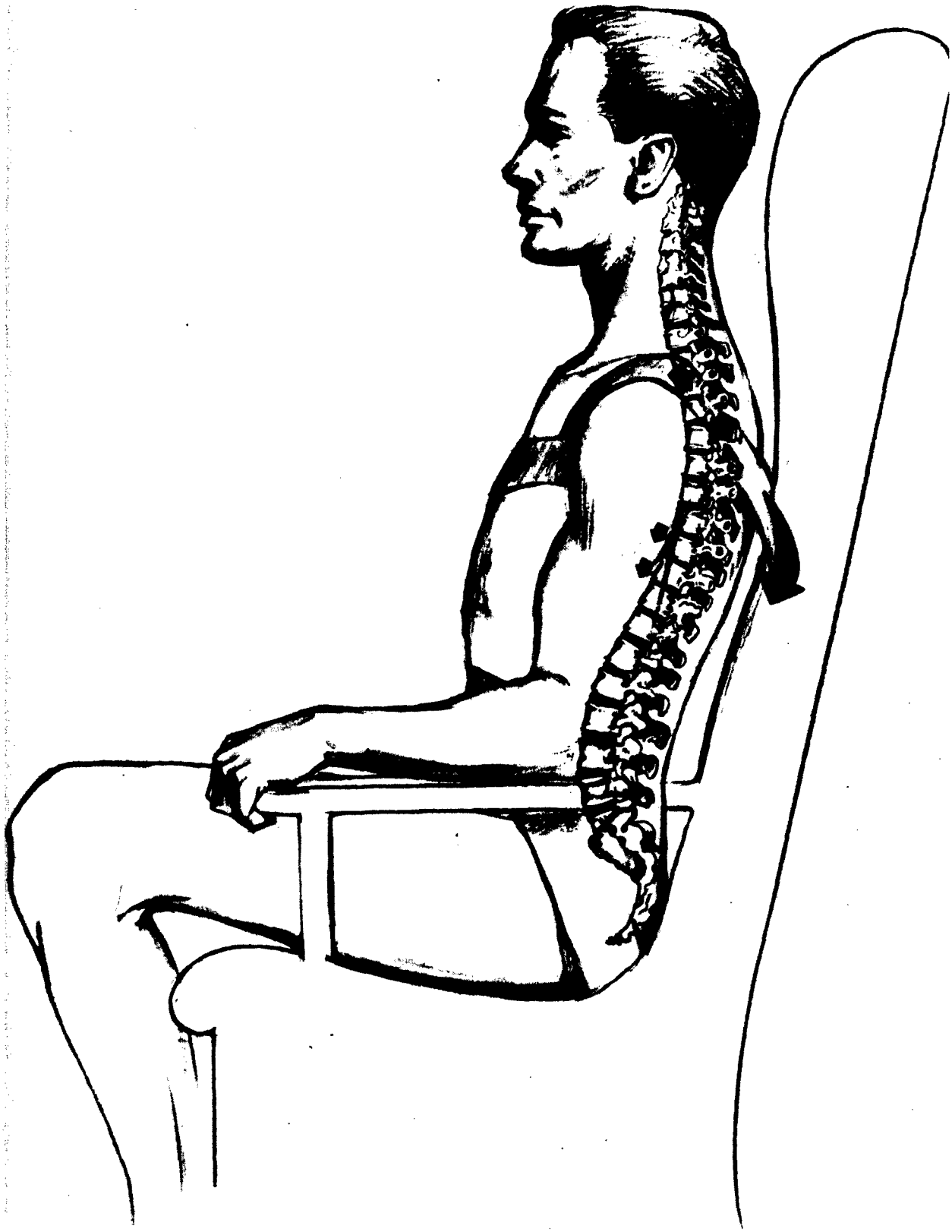


Figure 19. An improperly fitted shoulder harness will exert downward traction and preload the lower thoracic and lumbar vertebrae.

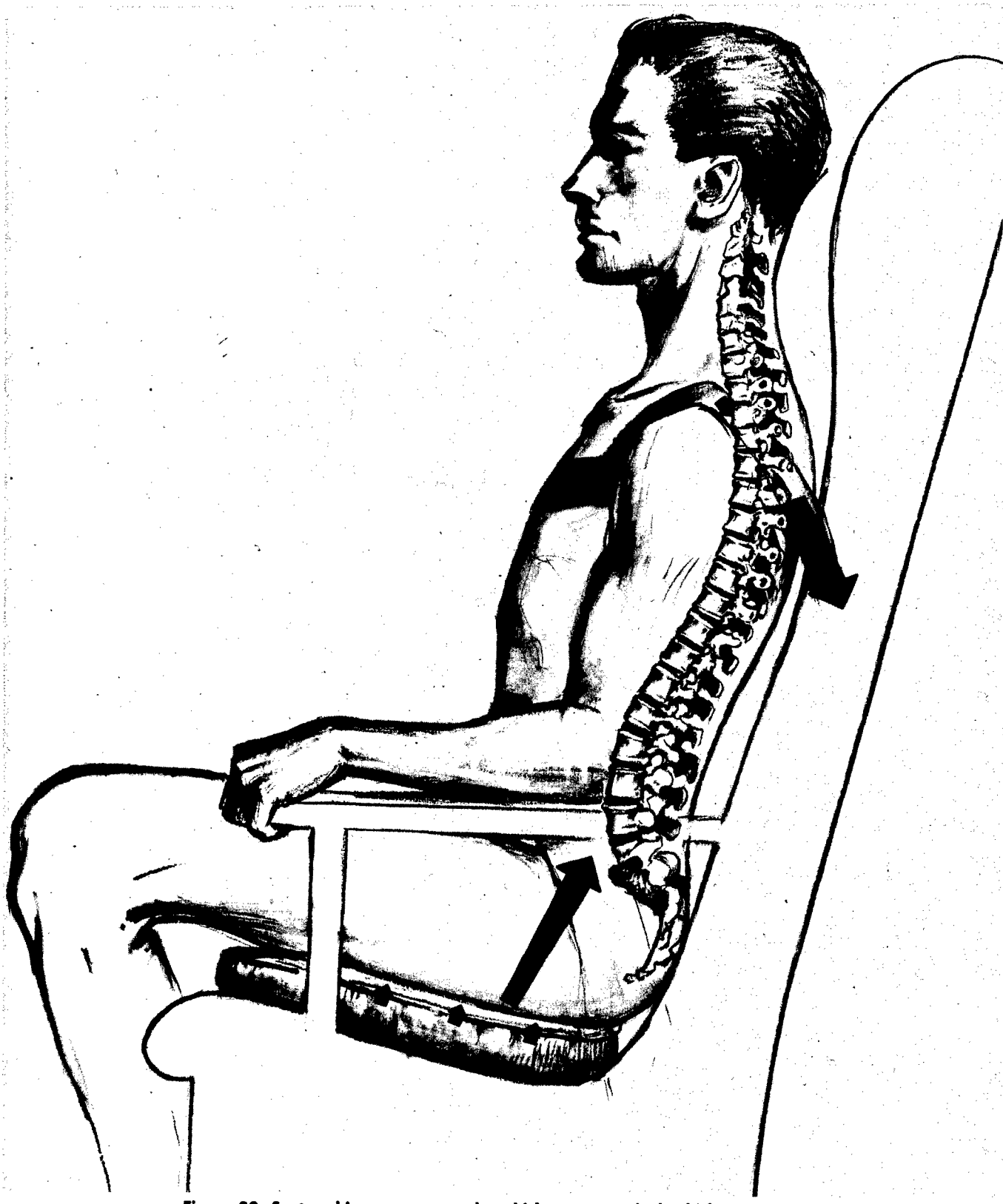


Figure 20. Seat cushions or seat packs which are excessively thick can force a crewmember upward into his shoulder harness which then combines with the offender to cause compressive preloading.

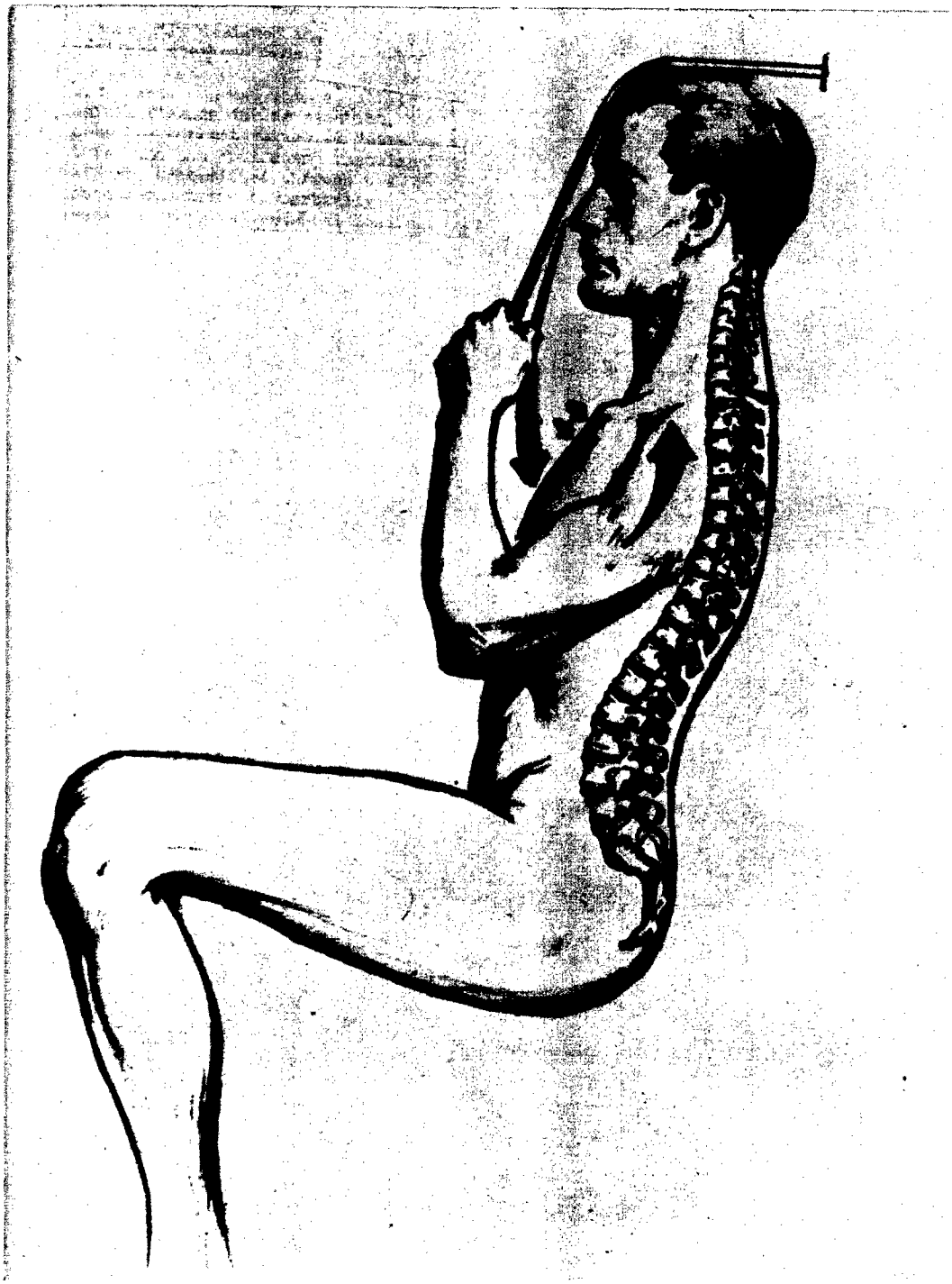


Figure 21. A correctly designed and properly utilized overhead mode of ejection initiation not only protects from windblast, it also limits spinal flexion during ejection, promotes correct vertebral "posture," and the "pulldown" muscular effort, transmitted through the shoulders, tends to partially ease compressive vertebral loading.

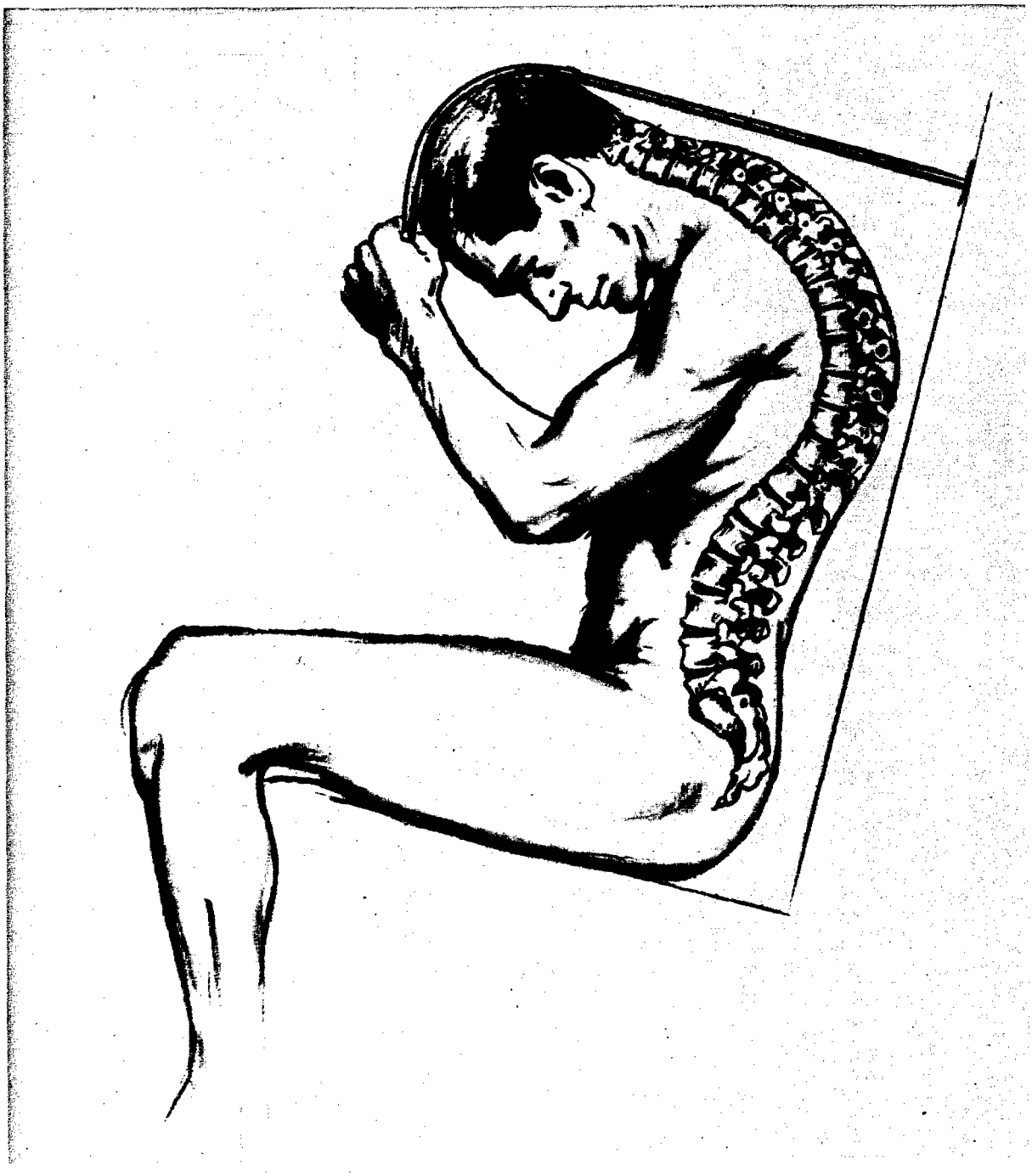


Figure 22. An improperly anchored or improperly utilized overhead mode of ejection-initiation may cause spinal flexion with its potential consequences.

are all important factors influencing safe ejections. To those subjectively and objectively involved with the increasingly complex problems of human escape systems, the above should represent guidelines.

It has been stated that the situation demanding ejection is life-threatening and vertebral injury would be preferred to fatality. In essence, this is true. However, with the biomedical knowledge, design ability and capability, and pilot caliber that is today available, a 20-25% incidence of vertebral fractures in nonfatal ejection sequences appears too high. This figure arouses more concern because it represents only those injuries detectable by routine x-ray of symptomatic individuals and does not include "silent" end-plate fractures. Additionally disconcerting is the realization that the factors described in this section as contributing to spinal injury are factors extrinsic to the ejection acceleration profile per se.

All of these factors would tend to indicate that in some areas of ejection seat design and use, more exacting application of presently available biodynamic knowledge should be exercised.

SECTION VI.

The Significance of Recognition, Management, and Prognosis of Ejection Vertebral Fractures

There is little doubt that a certain percentage of ejection vertebral injuries go undetected. Within this percentage are two primary groups: those who didn't have sufficiently detailed roentgenographic evaluation to document an existing fracture, and those who had fractures of a specific vertebral body documented but who had associated end-plate cracks which were too small to be concomitantly documented by the routine studies. Fortunately, as experience with this type of spinal column trauma is accrued and as diagnostic roentgenographic capabilities advance, the incidence of undetected or unrecognized spinal pathology diminishes. The reasons that minimal bony trauma has been missed in the past are varied but the following are probably two of the most common. In the first place, some fractures with minimal loss of vertebral height are not extremely painful. Such pain may not be immediately aggravating to an individual who has just undergone the mental and physical shock and strain of ejection. In addition, pain from other associated contusions, abrasions or more severe injuries may mask the nondisabling discomfort of minimal vertebral trauma. Even persistent minimal back stiffness may unwittingly be attributed to generalized whole body trauma without much subjective or objective attention to or concern for a residual "kink in the back." The other, probably common, reason for missed bony spinal trauma is the instance in which initial films are unremarkable and follow-up x-rays are not obtained. Many authors, including Rose and von Mentizingen (1930), have described patients with back complaints following acute trauma in whom original spinal x-rays were unrevealing. When x-rays were repeated from 2 to 4 months later, however, they revealed intervertebral disc substance which had finally broken through the damaged end-plate and had become ossified within the vertebral body.

Unfortunately, aerospace literature has come to equate, usually without qualification, negative x-rays with minor back injury. Instead of obtaining repeat films or special views, the tendency is to accept a negative film as proof for absence of bony deformation. Many authors, including Conwell (1952), have pointed out that in some instances in which early roentgenograms appear to be negative, subsequent films have revealed vertebral body collapse. A more reasonable approach is that if detailed repeat films made 6 months after ejection are unremarkable and the previously painful back is then asymptomatic, it is safe to assume that the individual had incurred a minor back injury. This is to say that his postejection back symptoms were due to muscular or fascial strain or tear which healed without sequelae.

Documentation of spinal fractures per se demands attention toward the anatomically distinct cephalic and caudal end-plates of the vertebral bodies. Minimal x-ray diagnostic ability is required to detect the compressive fracture vertebral height alteration, which in itself signifies end-plate rupture. The true challenge is to document the highly suspected end-plate fracture in the x-ray negative back. Only recently have clinicians come to appreciate the occurrence and significance of end-plate disruption.

In 1927, Schmorl (1927, 1928, 1931) and Putschar (1927 a, b), working independently, performed necropsy investigations of discs and vertebrae. In both series, rupture of nucleus pulposus through fractured end-plates was the most significant finding. Putschar found such lesions in 54 of 72 examined spines. Of the 54, 18 were microscopic and Putschar concluded that such lesions must have an end-plate displacement of at least 1 mm to be detected roentgenographically.

Schmorl found end-plate disruption and nuclear prolapse (Schmorl's nodes) in 38 percent of 3000 spines examined. Although he did not deny that acute back trauma could give rise to end-plate fracture and intraspongious nuclear transgression, he believed that most such lesions did not occur quite so abruptly. From the series Schmorl compiled between 1925 and 1930, he concluded that most of the end-plate damage was probably due to gradual degenerative changes, but that acute trauma probably also accounts for a portion of such injuries. Ubermuth (1929) also believes that accumulative degenerative changes cause the majority of nuclear displacement through ruptured end-plates and that acute trauma causes the smaller proportion of the cases. In using the term chronic degeneration, these authors meant gradual wear and tear subsequent to the repeated spinal loading which occurs from day to day. Degeneration in this sense is not to be interpreted as a primary disease entity.

Some authors believe that underlying embryologic defects in the end-plate, such as notocord remnants (Calve and Galland, 1930) or persistent vascular channels, more readily permit such injury. As mentioned earlier such defects do play a role. On the other hand, however, neither all Schmorl's nodes nor all end-plate fractures occur in areas of the end-plates where embryologic defects prevail. Along this same line, Perey (1957) has shown that the normal end-plate has a fairly symmetrical material strength distribution. Therefore, the majority of intervertebral disc ruptures that occur subsequent to either chronic degeneration or acute trauma disrupt end-plates which are anatomically normal. Since the enlightening investigations by Schmorl and Putschar, Wissing (1930) has closely reevaluated spinal x-rays from over 400 patients with various clinical problems. Evidence of past end-plate fracture was observed in 13.5% and he noted that all of those patients had previously complained of back pain following an accident. Also evaluated were almost 150 control spinal x-rays from apparently healthy individuals without complaints. Evidence of end-plate fracture was found in 16% of this group! Wissing has similarly confirmed such intraspongious disc herniation in patients who had no history of acute trauma. Indeed, the majority of end-plate fractures are probably degenerative in origin and for the most part painless in nature. However, this type of injury also occurs subsequent to acute trauma, in which event there is usually some associated pain.

Three important facts must be emphasized. First, routine spinal x-rays, which should be repeated at yearly intervals, should be on file for all flying personnel who may have to use ejection systems. The incidence of silent, degeneration-type end-plate disruption is sufficiently great that what is a normal series one year may be abnormal the next. Second, it should be realized that negative x-rays on a symptomatic postejection individual do not preclude bony injury. End-plate fracture must have fragment displacement greater than 1 mm in order to be visible on routine x-rays. Third, periodic follow-up x-rays should be mandatory for 6 months after ejection in symptomatic individuals and advisable in those without symptoms.

With diminished vertebral height subsequent to fracture, positive x-rays reveal variable characteristics of the fracture dependent upon the view obtained. Postural roentgenography can both exaggerate or minimize the damage so it is best to film the unloaded spine. Anteroposterior films usually reveal diminished height, but the slightly more desirable lateral views often additionally show a slight anterior displacement of the damaged vertebral body beyond its adjoining superior and inferior vertebral mates. Compressive fractures may be either transverse or vertical and are best delineated on lateral views. Recent fractures appear on x-ray as sharp, irregular lines, while the lateral margins of the vertebral body are usually smooth.

Throughout this report I have implied that routinely ordered anteroposterior and lateral spinal x-rays represent inadequate roentgenographic investigation of painful postejection spines. In addi-

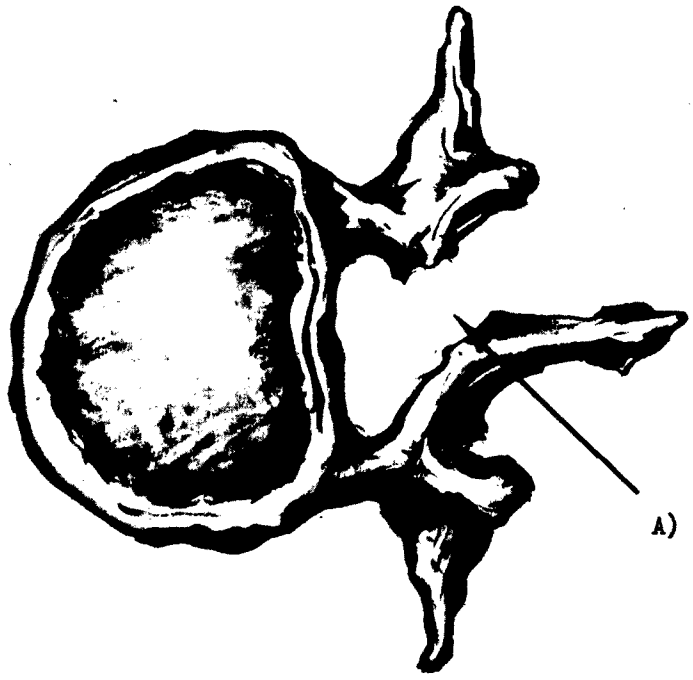
tion to these routinely obtained films several other more sophisticated x-ray views are available. Oblique views, which demonstrate the posterior vertebral joints in excellent detail, should be included in the roentgenographic evaluation of any spine in which there is a question of fracture. Besides anteroposterior, lateral, and oblique x-rays, three other roentgenographic studies should be mentioned. Discography, the injection of radio opaque material into the intervertebral discs, was originally described by Lindblom in 1948. This method of study is both reliable and highly accurate for delineating subtle end-plate fractures. Because of its high inherent risk, however, it has not for the most part been adopted as a safe diagnostic tool. Nonetheless, advocates of this procedure in the past, such as Cloward and Buzaid (1952), have documented reports of end-plate fractures, not demonstrable on routine spinal films, which were easily spotted using discography. Myelography was introduced and perfected in the early 1920's by such men as Dandy (1929), Spurling and Thompson (1944), Arnell (1948), and Knutson (1941). Either air or radiographic dye is injected into the spinal canal in order to visualize structures surrounding the spinal cord. Generally, this particular diagnostic tool is used to investigate primarily posterolateral disc prolapse and will not be extensively discussed here since it is not directly pertinent. An excellent report on the correlation between myelographic findings, clinical symptoms, and operative findings has been carried out by Friberg and Hult (1953). The best, safest, most readily available and accurate roentgenographic technique for delineating vertebral fractures is that of laminography or tomography. Instead of a single composite view of a particular structure, this technique permits visual detail at a particular depth. A spine subjected to lateral laminography is longitudinally sliced into a number of sections. The extensive and detailed spinal column tomographic studies of Weinbren (1954) have confirmed a relatively high incidence of unsuspected end-plate fractures associated with compression of different vertebral bodies. All individuals suspected of having vertebral fracture should receive tomographic evaluation.

Anyone associated with the biodynamic problem of spinal trauma should be aware of the extent to which congenital structural anomalies occur in the vertebral column. Breck and his associates (Lewin, 1955), in evaluating the spines of 450 applicants for heavy work, noted x-ray evidence of some type of anomaly in 31%. Although congenital defects may result in weakness of the spinal column, the structural anomaly may evade discovery until put to the test of chronic stress or moderate acute trauma. Indeed, Steindler (1955) has stated that "backs endowed with anatomical variations are inherently weak by virtue of natural restrictions of normal motion."

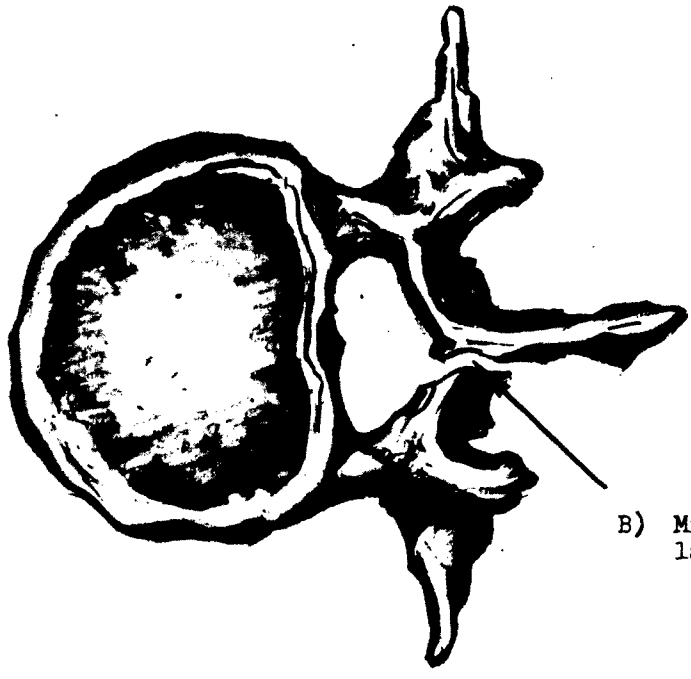
The two most common structural aberrations are spina bifida occulta and sacralization of lumbar vertebrae, both of which are illustrated in *figures 23* and *24*. Spina bifida, resulting from incomplete closure of the vertebral laminae and spinous processes, is probably most common at the fifth lumbar and first two sacral segments. Approximately 6% of the population has such a defect of L5, whereas the first two sacral segments are affected in about 11%.

Brailsford (1955) noted unilateral sacralization of L5 in 34 of 3000 individuals studied by spinal x-rays. In the same series 47 individuals had bilateral sacralization. People with spina bifida are usually asymptomatic and only about 0.1% of individuals with sacralization complain of pain. It is interesting that the individuals with sacralization who are symptomatic are usually those with a unilateral defect, whereas those with bilateral sacralization remain, for the most part, without complaint. To what, if any, extent these common types of defect limit tolerance to acceleration, is debatable.

In Hirsch and Nachemson's series of postejection fractures (1963), vertebrae stigmatized with such defects were found to be undamaged in the same vertebral columns that contained compressed vertebral bodies. These two, primarily posterior arch defects, probably do not significantly alter



A) Major portion of lamina congenitally absent.



B) Minimal but definite laminar defect.

Figure 23. Spina Bifida Occulta.

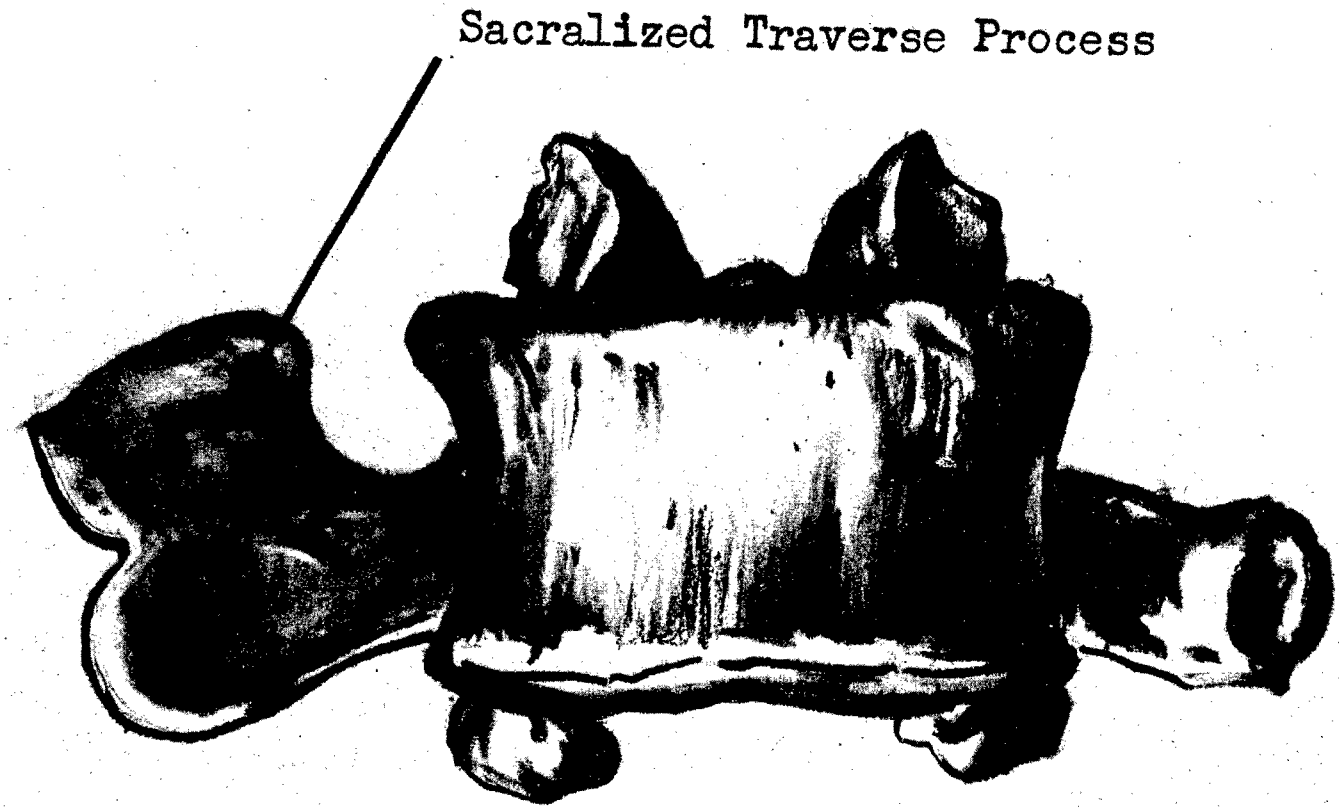


Figure 24. Sacrilization of Lumbar Vertebrae.

vertebral body structural strength. If they are noted on preflying status spinal films in an asymptomatic individual, no great concern should arise. On the other hand, when one congential defect is present, others are usually present also, though perhaps subtly obscured. For this reason it is probably best to "think twice" before allowing individuals who have demonstrable defects associated with even remote back complaints to become part of a pilot population. When vertebral end-plate fractures occur at acceleration levels below what has been termed "safe," congenital defects were probably present in the end-plate. Even if preflight laminograms were routinely made, which is neither practical nor reasonable, the yield of positive end-plate findings would be extremely low. The importance of laminography lies in its ability to document acute end-plate fractures so that proper treatment may be instituted to minimize potentially disabling future sequelae.

In summary, congenital structural aberration does occur. In the majority of instances it is asymptomatic. When, however, it is discovered in a pilot or potential pilot, it should be documented and the involved spine more thoroughly studied for further pathology. If oblique and laminographic x-rays yield no further evidence of defects, nothing more need be done. If associated defects are discovered, recommendations should be individualized.

An often disproved clinical dictum states that any injury of significance is usually symptomatic. It is now recognized that one of the disquieting features of minimal vertebral compression may be the paucity of acute symptoms. When present, however, pain, local tenderness, and variable disability are the usual heralding symptoms. Point tenderness over the precise site of the injury is probably the most frequently positive sign. Symptoms are not always immediately present and most individuals with vertebral fracture will soon assume a protective, flexed attitude; be sensitive to movement; and be unwilling to extend the spine. Although the degree of symptomatology serves as a rough index of the extent of injury, it by no means represents an accute guideline. Thus, an individual with minimal compression of two or three vertebral bodies may have relatively little discomfort whereas another individual with a solitary end-plate fracture may appear alarmingly uncomfortable. Whatever the degree of bony injury, spinal function should assume the primary role in spinal fracture rehabilitation, with vertebral form assuming relatively less importance.

Nicholl, in 1948, was one of the first to advance functional treatment of stabile compression fractures. Until then it was customary to be concerned primarily about the vertebral body deformation and to pay little attention to possible associated disc trauma or malposition of the intervertebral joints. It is now fairly well recognized that restoration of the form of the vertebral body does not always assure restoration of normal function. Complete bedrest immediately after any spinal injury is suspected, or documented as vertebral fracture, minimizes muscle spasm and also facilitates early healing. Return of physiologic spinal function, the primary goal of treatment and rehabilitation, is often enhanced in degree by early hyperextension of the back. This may be accomplished either during bedrest or, in an ambulatory patient, by means of a special back brace. Depending upon the extent and severity of vertebral body compression, early hyperextension immobilization often hastens uncomplicated, functionally normal recovery. Following are some acceptable rules of thumb. Individuals with a minimum of symptoms and having little vertebral deformity can usually continue every day, nonstrenuous activity if they have been fitted with an appropriate back brace. Individuals with moderate to marked loss of vertebral height are usually best treated by bedrest with or without hyperextension; and individuals with severe discomfort and marked distortion of vertebral body configuration, particularly when more than one vertebra is fractured, are probably best treated with hyperextension bedrest. No matter what the spe-

cific course of action; if disability is to be minimal or nonexistent, rapid return to functional activity is of the utmost importance. Hirsch and Nachemson (1963) reported 55 pilots who had complete spinal x-rays subsequent to catapult ejection. The 13 who had "unsuspected" vertebral fractures were back on flying status after an average convalescence of 2 months.

In any vertebral fracture injury the long-term prognosis is guarded. Factors determining the final outcome are the age and condition of the person incurring the fracture, the type and extent of the fracture, the presence and degree of associated injuries, the length of time that elapses both between injury and initiation of treatment and between injury and initiation of rehabilitation efforts, and finally the psychological makeup of the injured individual. It should also be mentioned that, as Landoff (1953) has demonstrated, there is a definite difference in prognosis dependent upon whether or not discs have been damaged. Although the ejection literature is almost void of reports on documented disc injury, end-plate fracture is known usually to result in intraspongious herniation of disc material. Since most symptomatic disc lesions result from pressure on nerve roots subsequent to posterolateral prolapse, many times central nuclear prolapse through the end-plate results in minimal symptoms. If disc-dependent difficulty does arise, it is probably due to more extensive disc trauma than isolated intravertebral body herniation. In Landoff's series (1934) of spinal trauma patients with fractures who did not have associated primary disc injury, all patients fully recovered, while those with associated disc injuries recovered completely in 55 to 75% of the cases depending upon other aspects of the fracture. Writers such as Olin (1939) and Hellner (1930) believed that disc lesions occur in 75 to 80% of all vertebral fractures. Such lesions are probably tears in the annulus for the most part. However, depending upon the degree of vertebral body disruption, all gradations of disc trauma from annular tears to nuclear rupture are possible. The frequent postejection pain and disability out of proportion to minimally observed bony damage is probably partially related to such associated disc trauma.

Reports of long-term follow-up on individuals who incurred ejection vertebral fractures are not readily available if they exist. A follow-up survey of the ejected population, though difficult to perform, could prove enlightening. In the cases of spinal fractures in the civilian population, which admittedly are subject to the overtones of post-industrial-injury malingering and post-auto-accident lawsuits, the outlook has in the past been rather dismal. In a series of 100 cases of acute fracture of spinal vertebrae without spinal cord injury, Conwell (1952) found a 23.5% incidence of permanent total disability. Five of the 62 patients from this group who had sustained industrial fractures were rated 100% permanent total disability, 12 who averaged 65% total disability were unable to return to industrial work of any kind, 20 resumed light industrial jobs and 25 returned to full duty without any permanent disability. Although the Armed Forces does grant disability for disease entities and incapacitating injuries that arise or occur during active duty, the problem of military ejection injury sequelae is primarily one of valuable manpower loss. Pilots or other crewmembers who incur disabling ejection spinal fractures are often superbly qualified, expensively trained individuals whose removal from flying status represents a significant loss. For this reason any rehabilitation effort that results in returning personnel to flying status is worth the overall time and effort. Only in recent months have the Armed Forces begun to appreciate the importance of well-guided, early rehabilitation. The uncomplicated compression fracture patients who are mobilized most rapidly and started early on properly directed hyperextension programs are those who are soon able to return to flying status. These are also the individuals in whom a minimum of immediate sequelae can be anticipated. Until long-term studies or surveys are available, it is impossible to accurately appraise the implications of even the most minor ejection back injuries. Ejected individuals who continue to have back symptomatology in the face of unrevealing comprehensive diagnostic studies are just as likely to develop long-term

sequelae as those with demonstrable lesions. Arthritic spinal changes are relatively common years after both back injuries in which fracture was proved and documented, and back injuries which were originally symptomatic but "x-ray negative." Symptoms of spinal nerve root compression have developed subsequent to postejection back complaints in individuals whose roentgenograms were initially interpreted as normal. In such instances an extension of an already present annular tear probably occurred during ejection with or without an undetected end-plate fracture. With continued everyday stress and strain after the postejection back complaints diminished or resolved, posterolateral disc prolapse finally occurs. The fact that it would probably have eventually occurred without the superimposed trauma of ejection is a moot point. The fact to be emphasized is that in a reasonable individual who has experienced spinal trauma, *a painful back is an injured back.*

In conclusion, the following must be recognized by all who in any way deal with the man-system components involved in escape from disabled aircraft. Ejection spinal trauma is not always a benign, short-term affliction with minimal sequelae. The implications of any vertebral trauma potentially portend physical disability, mental anguish, financial degradation, and valuable manpower sacrifice.

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13. ABSTRACT

Vertebral compression represents a significant percentage of the morbidity associated with upward ejection. Vertebral and intervertebral structure reacts to and is sometimes irreversibly altered by ejection acceleration. Design and material properties of the normal vertebral column are sufficiently constant that when structural characteristics are defined and acceleration profiles known, prediction of failure may be made. Compressive load analyses of vertebra-disc complexes have demonstrated that the vertebral end-plates are the initially failing structures of the spinal column. From experimental data on vertebral breaking-loads, acceptably accurate probability-of-injury curves for static loading have been generated. These data together with data describing the dynamic response characteristics of the human body permit calculation of the probability-of-injury for dynamic loading produced by exposure to impact accelerations. As an aid to the designer of ejection systems, application of these concepts should refine the estimate of "safe" acceleration profiles and minimize the risk of irreversible vertebral deformation.

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